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GASTRIC NEUROSES.

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There is, perhaps, no single word in medical terminology that has been made to cover so many deficiencies in learning and diagnostic acumen as the term "neurosis." For years it has been accepted as a full explanation of innumerable morbid phenomena and processes beyond the range of contemporary medical knowledge; and to no class of disease has it been more freely applied than to certain forms of disease of the stomach.

The very complexity of the classification and description of gastric neuroses in the text-books, the diversity of opinion concerning their causation, and the innumerable modes of treatment suggested by various authors lead us at once to question the supposed facts concerning them. The use of many drugs for a single condition leads us to doubt the value of any one; and irreconcilable differences of opinion on theoretical questions in medicine and surgery lead us to doubt the statement of any of the disputants. There can be no doubt, however, that there are certain morbid gastric conditions which have been and are still classified as neuroses, and that the surgeon as well as the medical practitioner is often called upon to confirm a diagnosis or suggest a treatment. It will be my purpose, then, to consider these so-called gastric neuroses, especially in so far as they concern the surgeon. Those manifestations of gastric disturbance primarily psychic in nature or associated with grave organic nervous lesions evidently do not come within the scope of such a discussion. We may discuss them, granting their

existence, but realizing their non-importance as regards the question at issue.

What concerns us particularly is that set of gastric symptoms classed grossly as "nervous dyspepsia." Under this general term have been grouped the most diverse symptom-complexes, with, as a rule, but little understanding of the underlying principles of the case. It is true beyond a doubt that there are certain disturbances in the gastric function, motor, secretory, and sensory, for which we can, by the minutest examination, find no organic basis. Besides grouping them into these three classes, we may also classify them as conditions of irritation or depression. Thus, gastralgia, nausea, and gastric hyperesthesia are prominent types of sensory disturbances; hyperchlorhydria and hypersecretion are well-known types of secretory disturbances; while atony, pylorospasm, and pyloric insufficiency represent well-known varieties of motor disturbance. Needless to say, motor, sensory, and secretory aberrations may all be combined in a given case, and it is by various combinations that the different types of so-called "nervous dyspepsia" are produced.

When we speak of any one or more of these conditions as a neurosis, we exclude by that very term the presence of an organic stomach lesion to account for the symptoms, or the existence of a lesion beyond the stomach which gives rise to them reflexly. Accepting them as neuroses, what is their frequency of occurrence, how may they be correctly diagnosticated and treated, and in what way do they reach the borderline of surgical practice?

As a fundamental principle we can safely state that a gastric neurosis without other neuroses or neurasthenic conditions is a most rare thing. In practically every case in which we finally conclude that the condition is a true neurosis it will be found that it is but one of a train of neuroses affecting the most diverse body functions and most widely separated organs. Careful examination may often be necessary to bring this fact to light. The gastric symptoms may so overshadow all others that attention is directed only to them. We would expect to find these neurotic or neurasthenic conditions most frequently in females, in the third and fourth decades of life. The picture that would present itself at once to the mind is that of a more or less emaciated, weak, and anemic patient. Often this may be realized, yet how often do we find neurasthenic manifestations, gastric and other, in the well nourished, and even robust, in persons of either sex, and in those in whom they might least be expected. Thus, for example, nervous gastric manifestations are not uncommon in farmers, a class in whom we would least expect to find them.

In what order of frequency do we find the symptoms? Of the sensory disturbances we find more or less anorexia or at least capriciousness of appetite in almost every case. It is such a constant

symptom in both organic and functional stomach diseases that in itself it is of but little value. Practically every sufferer from every form of gastric disease, real or imagined, complains of it at one time or another, and, as I have stated, the universality of the symptom renders its diagnostic value small. True gastralgia I have found very rare. It has been my experience that it is not a disease in itself, but a manifestation of disease of a definite organic nature, be it in the stomach, the central nervous system, or elsewhere. To this I will return later in speaking of its diagnosis.

Of the secretory disturbances, hyperchlorhydria is by far the most important. It is far more frequent than hypersecretion or gastrosuccorrhœa, which in its true form is indeed very rare, or than deficiency in hydrochloric acid in the gastric juice. Immediately there arises the question as to our ability correctly to diagnosticate such a condition of hyperchlorhydria, even if it be present. In spite of the vast amount of research work done upon the chemistry of the gastric contents and secretions, it cannot but be admitted that our knowledge concerning them is vague to a degree, and that our methods of examination are extremely crude. The normal variations of the amount of hydrochloric acid in the stomach contents and secretion at various ages and various stages of digestion, the individual deviations from a fixed standard, are very great. The normal amount for one person may be deficient or excessive for another. From a period of comparative confidence in our examinations of the chemistry of the stomach we have come to one of doubt and uncertainty. Where once an examination of the stomach contents was supposed to clinch the diagnosis in most instances, it is now generally considered of secondary value. My own experience with gastric analyses has been most unsatisfactory. Where once I had hoped that they would lead us to the possibility of definite diagnoses of ulcer, carcinoma, and secretory disturbances unaccompanied by organic lesions, I have now almost come to the conclusion that a gastric analysis is too often of but little help to a diagnosis. Yet extreme cases of hyperchlorhydria can be so diagnosticated, and do at times occur in the absence of anything that would seem to account for the condition.

As will be seen later, sensory and secretory disturbances of the gastric function are of import to the surgeon from a diagnostic point only. As we enter the field of motor disturbances we approach the scope of surgical treatment as well as diagnosis. Vomiting, eructations, and colic are, as a rule, but symptomatic. We may have apparently causeless, often repeated, attacks of vomiting, or vomiting of special articles of diet, in which operation or autopsy fails entirely to give us even a clue to the causation. In a recent article by a prominent surgeon published in the *Annals of Surgery*, a number of such cases are cited, and the author has apparently had brilliant results from exploratory operation, made, however,

upon false diagnoses. He even goes so far as to recommend operation for the cure of such a neurosis—an extremely radical and, to my mind, untenable position. A patient upon whom a gastroenterostomy was performed not only had no relief of symptoms but was much injured thereby.

Atony of the stomach also cannot at times be considered as anything but a neurosis, and its treatment falls fully as much within the province of the surgeon as of the internist. In the diagnosis of this condition we are again confronted by the lack of exactness of our methods of examination and the difficulty of fixing a standard with wide enough limitations to include all normal cases and yet of sufficient definiteness to be a standard.

Finally, we have that vague group of symptoms, sensory, motor, and secretory combined, which, in the absence of any definite or tangible demarcation, has been called "nervous dyspepsia." It includes definite feelings of distress, pain or heaviness in the epigastric region, eructations, anorexia, gastric torpor rather than marked atony, intervals of excess of acid secretion, and an associated intestinal derangement, with almost invariable constipation. This is the chronic picture of "nervous dyspepsia." It may entirely simulate a chronic gastric catarrh. It may include few or many symptoms, it may be a grave menace to the health of the patient, or merely a source of continual annoyance. It is the bane of both patient and physician; a long, slow, ever-increasing load of discomfort to the sufferer. Probably this may be due to the neuroses at times; as I hope to show later, it usually is not. Granted, even, that it may be, the name "nervous dyspepsia" has been overworked almost more than any single phrase, not even excepting the "uric acid diathesis."

It will be seen, then, that a gastric neurosis is a thing of infinite variations. It changes its symptoms from week to week—one might almost say from day to day. It may show itself in a complex group of symptoms or in the persistence of one particular inexplicable annoying condition.

The diagnosis of a gastric neurosis is probably the most difficult of all to make. The methods of examination of the stomach which are definite may almost be confined to an estimation of its size by percussion and a determination of its capacity. I have already expressed my opinion, based on personal experience, of the methods in use for determining the gastric motility and secretory power; as a whole they are most unsatisfactory.

The most important features in the diagnosis of any gastric neurosis is the eliciting of a careful history, which will show us the general neurasthenic condition of the patient. His ocular or cardiac symptoms may give us a clue. The presence of a manifest general nervous breakdown with an undoubted neurasthenia would at once predispose us to consider, at least, any gastric symptoms

present as but local signs of a general process. Yet such local manifestations are often found in patients in whom we would least expect an asthenia of the nervous system as a whole. Then, again, this run-down condition may be a secondary neurasthenia, due to a primary lesion which underlies both it, indirectly, and the primary condition of the stomach most directly. Thus, a patient with a latent but not symptomless gastric ulcer would soon show gastric symptoms, which might be considered nervous in origin, as well as a general neurasthenic condition due to his sufferings.

The variability in gastric symptoms would again point to a neurosis. A patient with a definite organic lesion of the stomach has a cycle of more or less definite symptom-complexes, progressively going from bad to worse. The neurasthenic, on the other hand, shows with every fluctuation of his general condition a corresponding change in the gastric status. Yet how difficult the diagnosis may be can only be appreciated by those who have had many stomach cases under observation.

A gastric ulcer, for instance, may manifest itself in any number of ways. If, for instance, the pain is the most prominent symptom, and vomiting is absent, as it is in a number of instances, how apt one would be to attribute the pain to a functional condition, and to call it a true gastralgia. Treatment might relieve it somewhat, but the diagnosis would remain doubtful until some acute manifestation of the ulcer brought to light the true condition of affairs. Then, again, an ulcer of another type might have as its cardinal symptom the hyperchlorhydria so often found as an accompaniment of ulcer. The physician's attention would first be directed to the excess of acid, and the absence of great pain and perhaps of vomiting would make the false clinical picture of a severe secretory obstruction, with its concomitant train of evil symptoms. An ulcer, again, may show its presence primarily by the vomiting which it causes, yet be entirely without bleeding. How easy it would be to confound it with a purely functional vomiting. Again, it might remain entirely latent for long years and give rise only to that vague picture of "nervous dyspepsia" before mentioned. Almost as common as gastric ulcer, in the experience of some surgeons, is duodenal ulcer. Its symptoms even more frequently than those of the corresponding condition in the stomach fail of recognition. Many indeed of these cases are diagnosticated as "functional" or "nervous dyspepsia," and they are allowed to go on until perforation or profuse hemorrhage occurs, which, while it makes clear the diagnosis, shows us also that valuable time has been wasted and the patient allowed to lose his best chance of cure.

It is evident that text-books dealing with diseases of the abdominal organs must be rewritten, as the descriptions they now contain of the symptoms of diseases of these organs are of no moment at all in making an early diagnosis. Until these text-books are

rewritten on the lines of living pathology, they will remain useless as an aid in making an early diagnosis.

Carcinoma in its early stages also is much more often considered as a gastric catarrh or nervous dyspepsia than it is recognized. The physician of the past but too often has allowed these cases to go on to the stage of entire hopelessness before really making a correct diagnosis. Anorexia followed by the symptoms of a vague chronic gastritis or neurosis, when it occurs in a middle-aged person, is a condition which should excite our greatest apprehension, and be dismissed only after the most careful examination has been made—after the case, if obstinate, has come to operation. When the carcinoma early gives symptoms of obstruction, we have simulated the picture of chronic gastric atony, not because the stomach musculature is insufficient for its work, but because of a constantly increasing obstruction to the outflow of the stomach contents.

Not only do we know that such gross and evident conditions as typical ulcer and carcinoma of the stomach may give us symptoms of so various a nature as to correspond almost entirely with the old picture of nervous dyspepsia, but more recent clinical manifestations have directed our attention also to a less evident lesion of the stomach. I refer to that condition of puncture ulceration of the stomach mucosa with small early bleeding points, which may involve almost, if not quite all of the gastric mucosa. In the absence of the classical signs of ulcer, which we often have in this condition, the hyperchlorhydria present has often been mistaken for the main lesion. This is not to be wondered at, as it is only by surgical autopsies on the living that we have gained an accurate conception of the condition. Its etiology, again, is quite as obscure as that of many of the true neuroses.

Carcinoma and ulcer may not only lead to a simulation of the secretory and sensory manifestations of gastric neuroses, but also of the motor neuroses. The resemblances between the evidence of a beginning pyloric obstruction and a marked gastric atony I have already mentioned. Various varieties of vomiting occur in both these disorders, and also in those of less marked pathological changes, such as chronic follicular gastritis or minute ulcerations of the mucosa. The vomiting in these conditions, and in carcinoma at the outset of the disease, is often not to be distinguished from the purely nervous types of recurrent vomiting described by many authors.

It has always been my opinion that in very many of the cases of vomiting regarded as primary neuroses we have really a symptom only of some lesion in or outside of the stomach which, for some reason or other, we have been unable to determine. Vomiting as a pure motor neurosis is regarded nowadays as far less frequent than it was thirty years ago, yet that we occasionally see it does not

permit of contradiction. The following case was to my mind one of gastric neurosis, a case in which surgical treatment was ill-advised:

Mrs. ——, aged twenty-nine years. Family history negative. Menstruation began at sixteen years, has always been regular; considerable pain. Has one child, eight years old. Never any miscarriages. Before having been operated on, six years ago, for perineal laceration (at which time there was also a curettement, shortening of round ligaments, and removal of small cysts from the ovaries), the patient had been subject to sick stomach and vomiting —in fact, difficulty in keeping anything on the stomach. Vomiting persisted, with nausea and great gastric distress, coming on soon after eating. Six months following the first operation a second operation was done (removal of both tubes and ovaries). Two years ago a gastro-enterostomy, and six months later an entero-enterostomy was done. The patient now came under my care, with a request that I operate on her. The symptoms complained of were much the same as originally: vomiting, the patient keeping nothing on the stomach, vomiting soon after eating.

Physical examination demonstrated nothing other than a movable right kidney. After having the patient under observation some days, making the usual tests which are done at the present time, I determined that she had a neurosis, and had had from the start; also, that I would operate on her with the idea of restoring the abdominal anatomy to its normal condition, minus the tissues, in the shape of intestine, necessary to remove. I opened the abdomen and excised the bowel included in the entero-enterostomy and gastro-enterostomy, uniting the jejunum to the terminal portion of the duodenum, and closing the opening in the stomach where the gastro-enterostomy had been done. The patient made an uneventful recovery.

The effect of this operation was to my mind purely psychical, and the present condition of the patient is due to the mental impression made by it. I found no obstruction at either the gastro-enterostomy or entero-enterostomy openings.

Our careful studies of case histories and minute observation of living pathology have convinced us that causes previously overlooked or considered insufficient fully suffice to account for extremely grave symptoms. The impossibility of diagnosticating a nervous dyspepsia with some slight catarrhal gastritis is manifest. It can be safely asserted that, while all sufferers from neurasthenia do not have gastric symptoms, all sufferers from chronic dyspeptic symptoms, from whatever cause, soon become neurasthenics.

Hitherto I have only spoken of the resemblance between so-called gastric neuroses and the symptomatology of organic gastric lesions. It will be evident that we must often be deceived in diagnosticating the presence of a neurosis, that in many instances there is in a supposed neurosis but the beginning of a serious organic

disease. Yet not only in the stomach do we find conditions often unrecognized, to the symptoms of which the older clinicians often applied the term neurosis, but there are a host of lesions of other abdominal viscera which deceive us in the same way.

We are coming to recognize more clearly every year the fact that the various organs concerned in digestion form a system correlated in every part. The stomach, intestines, biliary system, and even the appendix, the latter often described as functionless, do their work regularly, in orderly sequence, to accomplish the preparation of food for assimilation. Earlier brilliant researches in the functions of any one viscus often placed before us the picture of digestion as a series of related but almost independent processes. As a result of this conception the influence of one part of the system on all the others was but too often lost sight of.

The biliary system will illustrate my meaning to perfection. A patient who suffers from gallstones with stomach pains of an atypical variety will furnish the exact picture of so-called "idiopathic" gastralgia. All the other symptoms of gallstone disease may be absent, and the pain is attributed to the stomach neurosis when it really has a basis in the very solid fact of biliary calculi. During the attack of pain a transient hyperchlorhydria is not uncommon, and our very effort at diagnostic refinement by means of the stomach tube may lead us into still greater error.

Pericholecystic and peripyloric adhesions are very commonly found in association with bile-duct lesions of long standing. When they encroach upon the stomach or duodenum they often cause symptoms entirely gastric. An indistinct or vague history, pointing to the primary lesion is often passed over in a cursory history and we are at once led to believe that we must look for the seat of disease in the stomach. Indeed, actual pyloric obstruction due to pericholecystic adhesions is not unknown. In these instances the slow and long-continued course of the illness puts a diagnosis of carcinoma out of the question, the "atony" of the stomach is much in evidence, and we may almost be pardoned for an error in the recognition of the true lesion.

Simple gallstone disease is often described in text-books as being without symptoms. We read of postmortem statistics in which these cases predominate, and it has been asserted that nine-tenths of all gallstone cases have no symptoms. This is certainly not true. Almost every case of such a "symptomless" group would, if the history were minutely scrutinized, give us the story of a patient afflicted for months, years, even decades, by "chronic dyspepsia." We have learned that while the great classical symptoms of gallstones are often absent, almost every case will show marked evidences of impaired digestion. These patients complain of belching, anorexia, vague epigastric distress and flatulence, etc. They are the very cases that so many practitioners put on such liberal diet of

pepsin, sodium bicarbonate, diastase, and remedies of a like nature, that it should excite our wonder that after a few months they are able to digest anything. Mr. Moynihan, of Leeds, in his lecture on "Inaugural Symptoms," has called attention very strongly to the fact that gallstones are not, as is often stated, without symptoms. He has aptly cited the term "gall-bladder dyspepsia" as accurately describing the digestive symptoms which precede by months or years the late or so-called "classical" signs of gallstones. Our medical and surgical authorities have commonly neglected to recognize or describe these phenomena.

Turning to another portion of the digestive tract, let us consider the appendix. Chronic disease of this little organ, so long erroneously termed rudimentary and functionless, gives us many cases of chronic dyspepsia. In part reflexly (pylorospasm), and in part by direct influence with the cecal and appendical functions by kinks, adhesions, and malpositions of the organ, the picture of a chronic intestinal indigestion develops, and is very hard to distinguish from dyspepsia having its primary seat in the stomach. We must not fail to recognize the fact that malpositions or inflammations of the appendix, as found in chronic appendicitis, may cause symptoms far removed from the organ by direct action upon the sympathetic nerve fibers which ramify in every part of the abdominal cavity. Only within the last few days I have performed two appendectomies for marked chronic appendicitis in patients in whom the entire pain and distress were located in the epigastrium, not even referred to the right iliac fossa (typical instances of pylorospasm). I have had similar cases many times. Each recurrent attack of pain is referred to as "colic," and the true origin of the seizure is but too often overlooked. The marked constipation and flatus which in so many instances is the accompaniment, cause, or result of a chronic appendicitis serve to render the patient's condition still more miserable. Medical treatment, or, indeed, any form of treatment, not directed to the removal of the cause of the patient's distress must of necessity be futile. It is true that we must be on our guard when diagnosticating such a dyspepsia as of appendical origin. The reverse of this dictum is also true—that in some instances an indigestion not arising in the appendix, but accompanied by constipation, with cecal distention and tenderness, may so simulate a chronic appendicitis that an appendectomy is performed. Any surgeon who has done much work in appendicitis can look back on not a few cases in which an appendectomy for a supposed chronic appendicitis, in the absence of a history of well-marked acute attacks, has failed to give the relief expected of it. Our internists and gastro-enterologists diagnosticate many cases of gastric, intestinal, and nervous dyspepsia. Hereafter they may save their patients much suffering and themselves much chagrin by more frequent diagnoses of gall-bladder dyspepsia, duodenal dyspepsia,

and appendiceal dyspepsia, and by advising prompt surgical interference. I have no doubt that such refinement of diagnosis is but the question of a short time.

I need but mention the gastric neuroses of various forms reflexly caused by renal lesions, such as movable kidney, lesions of the female pelvic organs, or of the male genito-urinary system, to call to mind a picture of disease difficult at times to diagnosticate, and extremely resistant to any form of treatment, but only indirectly within the province of the surgeon.

It is evident, then, that there are many conditions which directly or reflexly cause the occurrence of what is termed "nervous dyspepsia," and it is my firm belief that, as a rule, the origin of the symptoms can be found in some very definite lesion within or beyond the stomach.

Yet it must be admitted that there are certain cases for which, apparently, the term "nervous dyspepsia" must suffice as a diagnosis. When we have eliminated the possibility or probability of a fundamental lesion, and find a case in which there are the symptoms of dyspepsia, especially one in which the distress occurs only after the ingestion of food, but regardless of its quantity or quality, we may be sure that we have to deal with a true case of gastritis nervosa. The existence of the general symptoms of neurasthenia would make the assurance doubly sure.

What, then, is the attitude of the surgeon toward these cases? I think that it should be "hands off," with but very few exceptions. The surgeon's principal duty, as regards the true neuroses of the stomach, is to recognize them, to separate them from secondary dyspeptic conditions due to lesions which perhaps it is within his province to treat. I regard the proposition to operate on these cases for the mental effect upon their general neurasthenic or hysterical condition as unsafe, illogical, and as setting a most dangerous precedent. It is certainly a confession of weakness in our therapeutics, medicinal and psychic, to suggest such a course.

These cases belong to the internist and neurologist; whether the neurasthenia be primary or secondary to the stomach lesion, our treatment is the same. They are in need of rest, with general and special dietetic treatment. Removal of the primary mental cause is essential, and these patients demand the most careful management if their recovery is to be assured.

There is no exception perhaps to the general statement that gastric neuroses per se are not within the province of the surgeon. If we consider gastric atony and ptosis as really neuroses, when they are apparently primary, they form the exception. There are certain of these cases in which all medical and general treatment is unavailing, while a gastro-enterostomy promptly leads to recovery by furnishing the stomach with drainage, which by its own force it is unable to secure. In ptosis, I have never seen good results from the

various methods devised for the purpose of raising the stomach itself. I believe that gastro-enterostomy is the only logical procedure. This is true even in some instances in which the pylorus is entirely patulous. While I believe that an occlusion of the pylorus is the main indication for gastro-enterostomy, I do not think it is the only one.

In conclusion, I cannot do better than to repeat my warning against operative interference in neuroses of the stomach.

THE TREATMENT OF CHRONIC BRONCHITIS.

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Chronic bronchitis is due to so many causes that its successful treatment is a difficult problem and frequently baffles the most skilful therapeutist. If we are satisfied with treatment of symptoms, as is commonly done and as, of necessity, must frequently be done, a routine therapeutic procedure can easily be prescribed. But this cannot be satisfactory and, as a matter of fact, is not satisfactory. The patients most commonly give up consulting physicians and take such remedies as they choose, namely, some expectorant or opium, and the physician, after having prescribed all the routine remedies, finally tires of efforts which seem futile. It seems supererogatory to state that both causal and symptomatic treatment are necessary for successful treatment of any disease; it may be that the latter includes the former, but both should be applied when possible.

Causal treatment has not received sufficient consideration in chronic bronchitis, which can be explained by the fact that the etiology of chronic bronchitis is very complex and not always discoverable. As indicated by pathological anatomy, there are many forms as well as many stages in this disease; but in all forms there is some change which eventually produces loss of resistance and of elasticity in the elastic and muscular tissue of the bronchi. This may be due to a variety of causes acting upon the mucous membrane from without or from within the body, or upon some inherent weakness of the tissues themselves. For causal treatment the following may be taken into consideration.

1. *Local Causes.* Lesions in the nasopharynx; inhalation of irritating substances, mechanical, chemical, thermic; infections with Bacillus catarrhalis, the pus producers, Bacillus influenzæ, measles, pertussis, tuberculosis, syphilis; diseases of the pleura—pleural adhesions, chronic exudates; diseases of the lungs—asthma emphysema, chronic indurations, tumors, chronic enlargement of the bronchial glands; and malformations of the thorax.

2. *Remote Causes.* Diseases of the heart; diseases of the kidneys; arteriosclerosis; rickets; scrofula, tuberculosis; chronic intoxications (alcohol, gout, auto-intoxications); obesity; heredity; all influences preventing normal respiration; refrigeration (catching cold); and all general causes predisposing to infections.

No case of chronic bronchitis should be treated without considering its cause or causes; but it will be seen from this list that some of these causes act upon the bronchial mucous membrane from within, others from without, some in both ways. In chronic alcoholism the alcohol which is eliminated by the lungs is the chemical irritant which produces the bronchitis. In all those infectious diseases in which the microörganism is carried by the blood current and then localizes itself the production of chronic bronchitis is from within. On the other hand, inhalation of irritating substances is an example of the production of chronic bronchitis from without. In tuberculosis we may have a combination of both modes of infection, as the bacilli may be inhaled, may be developed in the bronchi by extension, by continuity or contiguity, or may be carried by the blood or lymph currents. But whichever way the bronchitis is produced, it is necessary to consider the existence of some form of predisposition. This can easily be demonstrated by observation; the same local cause produces an ordinary acute bronchitis in one subject, in another it is followed by a subacute bronchitis which is the precursor of a chronic bronchitis; or, an acute bronchitis disappears and leaves a local predisposition to infection, or becomes a locus minoris resistentiæ to irritations of any sort, so that eventually a chronic bronchitis develops. Clinically most cases of chronic bronchitis not due to circulatory trouble develop from acute or subacute attacks of bronchitis which recur. The latter are looked upon as "only a cold," and therefore are not considered of sufficient importance to require attention. It is at this stage of the disease that treatment is most important; it may be possible to remove the damage done to the bronchial tubes, or further injury to them may be prevented. In the latter sense we are acting prophylactically, which in this disease is of incalculable value.

Catching cold, which consists of infection plus predisposition, should be prevented. The local predispositions in the nasopharynx should be removed; anything which produces mouth breathing puts in abeyance the filtering functions of the nose, and in its turn makes excessive demands upon the protective functions of the lower sections of the respiratory tract. When there is no mouth breathing, but simply local loss of resistance in the mucous membrane of the nasopharynx, gargling with large quantities of water and cleansing the nose by douching with indifferent solutions twice a day is of the greatest value. For our purpose, it is indifferent how the microbes which produce the "cold" reach the bronchial mucous membrane, whether by continuity of tissue or by the blood route, provided they are

removed from the first place of infection. This is effectually done in the way just described, as I have repeatedly seen when it was followed conscientiously. In "catching cold" the general predisposition should also be considered. It is common practice to advise peculiar kind of clothing, dry feet, care in exposure to draughts of air or to changes in temperature. It is very difficult to follow these directions unless the patient considers the prevention of catching cold the principal object of life, and many so consider it. Moreover, if general resistance can be increased the procedure is more natural, more easily followed, and more effective. This is done by the prevention of faulty metabolism and the deterioration of general health. These being attended to, the most important prevention consists in that procedure which has been called Abhärtung by the Germans—hardening. Essentially, this process consists in reducing the reaction of the skin to thermal irritations by gradual education. In doing this we reduce vasomotor reaction in the skin, which is followed by contraction of bloodvessls, and, therefore, we prevent sudden hyperemia in internal organs. Hyperemia upon mucous membranes is one of the principal predisposing causes of infection; this is shown for bronchitis in chronic heart and kidney troubles; acute hyperemia being prevented, the local predisposition is removed. That this is the only way in which the various procedures applied for hardening act may be denied; that the procedure is valuable cannot be doubted. Hardening is accomplished by hydriatic measures, the cold sponge, douche, or plunge; it should be done daily, and it should be modified in accordance with individual indications and limitations.

The infectious nature of "catching cold" should not be forgotten. While we rely upon normal or increased resistance to destroy bacteria, neither of these is present in patients with chronic bronchitis, and it is safer that they be kept away from infection than be exposed to it. It is justly and properly recommended that these patients be kept in the house during rough weather, or that they seek milder climates. Rarely, however, is it advised to prevent infection by not coming into close contact with other people who have a cold. Most people are not able to stay in the house or to leave home for a great length of time, and, after all, when they do the one or the other they are largely preventing infection. When a house epidemic of colds starts, the patient with bronchitic tendencies, who has been left in the house, gets an attack of bronchitis; when an epidemic of colds starts anywhere, in Egypt, Algeria, or Southern California, provided these patients are exposed, the exposure is followed by an attack. All this is thoroughly understood for influenza, and this has done not a little to our understanding of common colds; certainly it is not asking too much to keep predisposed patients away from the possibility of infection.

Auto-infection is more common than is supposed. The bacteria

which are found in the nasopharynx are more or less readily disposed of, as has been shown. It is a much more difficult matter when they are retained in the bronchial tubes, producing exacerbations by temporary increase of virulence or diminution of local or general resistance, or simply cause constant irritation by their presence. The removal of these bacteria when they are in the nose or throat may be easy, as has been described; when they are in the bronchi it is difficult but not impossible.

Bacteria can be removed from the bronchial mucous membrane mechanically, or they may be destroyed. Everything which increases secretion from the mucous membrane removes the surface bacteria; therefore inhalations, medicines, and other measures may be tried. For the destruction of bacteria in bronchitis little has been done. Remedies may be applied directly by intratracheal injections, or indirectly, as such that are eliminated by the bronchial mucous membrane when administered by the mouth or otherwise. Either way is useful, but there is no certainty that bacteria are destroyed; that their number may be diminished is not denied, but from what we have seen this is not sufficient. So that other things, in the way of protective measures, must be found to destroy bacteria, or, at least, to prevent their multiplication. Destruction of bacteria can take place by general means by the introduction of peculiar substances whose properties are well understood (bacteriolysins, bacteriocidins, etc.,), but I do not know that they have ever been applied in the treatment in bronchitis. In a number of cases of streptococcic bronchitis, antistreptococcic serum has been valuable. It is much more to the purpose, at least at present, to destroy bacteria in this stage of the disease by improving local resistance, diminishing local predisposition, or increasing general resistance. A hyperemic mucous membrane has diminished local resistance and increased local predisposition; if the cause or causes are removed this disappears, bacteria cannot grow because their soil is rendered unfavorable and they are actively opposed in their growth. We are accordingly not astonished to find that digitalis has been successfully administered in chronic bronchitis. The thought has not come that it acts by relieving a cardiac cause. "Chronic bronchitis with profuse secretion," says the fifth edition of the U. S. Dispensatory (1904) "is the only inflammatory affection in which it (digitalis) may be advantageously used." As will be observed, it may be advantageously used because the disease is inflammatory, not because chronic myocardial insufficiency is relieved. General causes are of even greater importance in changing the local conditions; chronic intestinal auto-intoxication, gout, and alcoholism should always be considered and excluded. In a certain number of case there is hereditary local predisposition due to some defect of physical or biological qualities in the respiratory apparatus; it is possible to detect this in children and to reckon with it.

The effect of diseased lymphatics upon chronic bronchitis has attracted little attention. There are very few cases of chronic bronchitis in which some change is not found in the bronchial glands, usually a hyperplastic condition clinically marked by enlargement of the posterior cervical glands. The existence of enlarged bronchial glands is easily explained by the direct transportation of the causa irritans to them and its localization within them. The enlargement of the cervical glands because of changes in the bronchial glands not only depends upon the inability of the lymph to be discharged in its proper manner, but the cause of the bronchial enlargement may also be the cause of enlargement of the cervical glands. Hart has shown that in injecting India ink into an animal there is a retrograde transport of this substance from the thoracic duct during inspiration, affecting many glands, but also the bronchial and cervical glands. Whether the bronchial glands be hypertrophied or, as they become later in the disease, atrophic, the effect upon the cervical glands is the same in regard to enlargement except as to degree. Under all circumstances enlargement of bronchial glands produces a distinct effect upon the condition of the lungs and bronchial tubes, which is greater in the mucous membrane of the bronchi than in the lungs, because they are more remote and because the forces causing lymph flow in them are of a more passive nature than in the lungs, especially when they are diseased. The effect produced is that of stasis in the lymphatics, causing a moistening of the whole mucous membrane, a dilatation of lymph spaces and compression of blood-vessels, a copious transudation into the lumen of the tubes, and a condition most favorable to infection. Moreover, functional activity of the protective agencies of the bronchi is diminished, which is followed by the usual results. The treatment of these enlarged glands is frequently followed by excellent results. As a causal indication it should always be considered.

As in all diseases produced by microbes, reduced general resistance is followed by reduced local resistance. It will be seen, then, that reduction of general nutrition, reduction of nerve tone, and anemia act as predisposing factors whose removal by proper treatment is invaluable not only in the first stage of the disease, but also when it has become thoroughly established.

Climatic treatment acts in various ways. There can be no doubt that external temperature is of importance; a patient whose exacerbations come with cold weather may escape them provided he goes to a warm climate. But is it overlooking the mark if this is the only factor taken into consideration in advising change of climate. Send this same patient to a warm or hot climate in a place in which he is constantly forced to breathe dust-laden air, and the result will be far from favorable. Again, the latter or the former may be absent, and yet a change in climate will do no good. Change of climate acts causatively as well as symptomatically, which should always be

considered when it is recommended. The way in which change of climate acts beneficially is not so easily explained as would seem on first thought; besides those already mentioned, moisture and altitude must also be considered, and it follows that recommendation of a proper place for an individual is very difficult. Moreover, in recommending change of climate we are not only guided by the disease, but also by the adaptability of the patient to such a change or to any change.

In a certain number of cases the chronicity of the disease is evident from the beginning, but in the greater number it begins as a "winter cough," which gets worse from year to year and lasts longer and longer. In these cases of recurring bronchitis prophylactic treatment is of the greatest value and usually neglected. The attacks should be treated as carefully as possible, the patient should be kept in the house, in bed when there is fever, proper medication prescribed, and his general health considered. In many individuals these attacks are followed by reduction in weight and strength, so that convalescence is slow and predisposition to infection increases. It is in such patients that superalimentation is necessary; many of them do well on codliver oil. Sometimes these attacks are provoked by dust inhalation, especially in cities, and I have known several patients ward them off by wearing respiratory masks. As a rule, this is impracticable, but those in whom this etiology is established, whose occupation causes inhalation of irritating substances, and in those who cannot leave home it should be encouraged. The best preventive is permanent change of climate; next comes temporary change during the season when the bronchial attacks occur. When the irritant can be removed, as is the case in smokers who inhale the smoke, much can be expected for prevention of recurrence.

For one reason or another our patient has gone into the next stage of the disease, in which he is never entirely free of his bronchitis and in addition has exacerbations. He now has pathological changes in the bronchi which are permanent and progressive unless something can be done for him. It matters not what form of chronic bronchitis is considered, the hypertrophic or the atrophic, the inevitable final result is destruction of muscular and elastic tissue. In the hypertrophic form there is great cellular infiltration, which at first prevents normal muscular and elastic functional activity and finally destroys it. In the atrophic form there is substitution of fibrous tissue for the normal in mucosa and submucosa. In all the forms there is change in the mucous membrane: in its epithelial layer from incomplete to complete loss of cells to metaplasia, and in the lower layer infiltrative or atrophic changes. There is much congestion of the bloodvessels. Sooner or later all the tissues are changed, including cartilage, by infiltration and final atrophy. All these changes are followed by change of functional activity. In the epithelium the protective power of the ciliated cells is lost, small foreign bodies as well as

particles of mucous cannot be removed. In the elastic and muscular tissues the function of motion is interfered with or destroyed, and the material which collects upon the mucous membrane is not removed and remains as a constant irritant to produce renewed irritation and inflammation. The changes in the deeper layers cause weakening and loss of resistance to pressure which are followed by dilatation of the bronchial tubes. Not only do the bronchial tubes dilate because of expiratory efforts, such as coughing, but ordinary inspiration may at times be followed by dilatation. Because of the effect of diminished respiration, which occurs in chronic bronchitis, as well as destruction of bloodvessels, changes in the heart, especially hypertrophy of the right ventricle, occur.

As failure of removal of secretion is then so important, it is no wonder that this removal has been looked upon as the principal object of therapy. It is true that the usual ratiocination is not the one I have just applied, but is as follows: Secretion into the bronchial tubes produces coughing, the more secretion the more cough, the more cough the more annoyance, therefore, the secretion should be removed or the cough be checked. Those medicaments called expectorants have been employed for this purpose. They are directed to increasing fluidification of the expectoration, to irritation, so that increased efforts are made to propulsion, and to causing the secretion to be dried up. In chronic bronchitis it is rarely necessary to employ the second kind of expectorant, to which belong musk, ether, camphor, ammonium carbonate, alcohol, and others; certainly routine use of any of these drugs is contra-indicated.

In the first class there are nearly all the alkalies—ipecac, apomorphine, ammonium chloride, and pilocarpine. In the third group there are turpentine, benzoic acid, and creosote, their preparations and derivatives, copaiba, myrtol, eucalyptol, tar, and the astringents, such as lead or tannic acid, and belladonna and its preparations. The iodides are also valuable as expectorants, since they liquify the expectoration; but as they are eliminated by the bronchial mucous membrane and irritate it, they should be cautiously administered in chronic bronchitis. Beyond the general indications it is impossible to decide between the various members of each group, except by considering the possibility of unfavorable activity. In the bronchitis of Bright's disease we should be very careful to give only such remedies as do not irritate the kidneys. Aside from these considerations, our preferences are determined by actual experience: of the first group I use chloride of ammonium, ipecac, the alkalies, and pilocarpine; of the second class of expectorants, camphor and carbonate or anisate of ammonium; and of the other group I prefer turpentine, the benzoates, creosote and its preparations or derivatives, eucalpytol, and tar.

The same effects follow inhalation as are produced by medicines. There is some doubt of their modus operandi, but there is no doubt

of their efficacy. In order that they attain their maximum efficiency the fluid used should be as finely subdivided, vaporized, as possible, and its temperature not too far from the internal temperature of the body. The apparatus are of variable construction—inhaling masks, so called atomizers or nebulizers, inhalation cabinets or chambers. The choice depends more upon the kind of patient than upon the nature of the case. I have obtained excellent results by using two layers of a frequently washed old woollen petticoat as an inhaling apparatus, but manifestly that would not do if the petticoats are neither old nor of wool. By using the old fashioned croup kettle an inhalation chamber may be improvised. For the purpose of fluidifying the expectoration, inhalation of aqueous vapor of sodium bicarbonate or of Ems water; for diminishing the expectoration, turpentine, tar, creosote, eucalyptus, menthol, and tannic acid, can be recommended, applied in the same manner.

Intratracheal injections seem to be followed by good results. The fluid is injected into the trachea by means of a peculiarly constructed syringe; but to do this requires training. The following substances, dissolved in olive oil, have been applied in this manner: menthol, carbolic acid, guaiacol, iodoform, morphine. From what I have seen, this mode of treatment has been beneficial, but only for relief of symptoms, and this can be effected by simpler means in uncomplicated cases of chronic bronchitis, so that, at least in this form, it is not necessary except in advanced cases.

In ordinary cases postural treatment is the best for the removal of secretion from the bronchial tubes. In a normal bronchial tube the secretion is removed during the night by the ciliated cells; when secretion is increased this is no longer possible, and the patient must remove it by coughing—always in the morning, frequently at night as well. The secretion collects and calls the protective function of coughing into action. Because of our position in bed during sleep the bronchial secretion either remains stationary, when the body is level, or, when the head is higher than the trunk, gravity forces it away from the larynx; in either case collection of secretion takes place. If we change these positions so that the head is lower than the trunk, bronchial secretion neither stagnates nor does it move in the wrong direction, as the combined effect of ciliatic motion and gravity is sufficient to remove comparatively large quantities of secretion. This is accomplished when the patient sleeps with the foot of the bed elevated; in most cases this is sufficient; at times, depending upon the side and seat of the lesion, combinations of ventral or lateral posture may be necessary. The foot of the bed may be elevated by putting wooden blocks or bricks under it; whenever it is possible it is advisable to get beds which are so constructed that one end can be elevated. Long experience has taught me that the first elevation should not exceed two inches; later this may be increased to four or five inches. The combined postures are

obtained by the use of pillows or bolsters in the inclined bed; as a rule, they are not successfully carried out, but in a number of instances I have obtained good results. Whatever is done must be done gradually. Frequently the patient is unwilling to try a posture which is the opposite of the one he naturally takes to relieve his symptoms (cough, dyspnea). Moreover, it is very difficult for most people to sleep with the head lower than the trunk. When therefore a patient is found who sits up all night so that he can cough more effectually, it will not do to begin immediately with elevating the foot end of the bed. This is true also in dyspnœa and in very nervous people. When these conditions exist, I ask that the patient try with fewer pillows, then the horizontal posture, and finally the elevated one. When this is done there are few failures in the continuance of the treatment. After the patient has tried to carry out directions for a few nights, there is no further difficulty. As soon as the patients can pass the greater part of the night with feet elevated the symptoms begin to disappear. They cough less during the night, therefore sleep better; the morning cough is diminished and the amount of expectoration is lessened or disappears entirely. In a number of instances I have seen what may be called a cure, as all symptoms of the disease disappeared, but relapses occurred after a longer or shorter time, as could have been expected from our etiological conception of chronic bronchitis. This mode of treatment is applicable to all forms of chronic bronchitis in which the removal of expectoration is essential. It therefore is of no value in dry bronchitis. It cannot be expected to be followed by benefit in cases in which gravity does not assist; for instance, when elevation of the feet prevents the emptying of a dilated bronchus, then a combined posture may be of service.

In fœtid bronchitis intratracheal injections of iodoform in oil (0.4 per cent.), or inhalations of carbolic acid, equal parts of carbolic acid, alcohol, and glycerin, of thymol, 0.05 to 0.1 per cent. solution, of a 0.5 to 2.5 per cent. solution of oil of eucalpytus in alcohol and water, are useful. Internally A. Fraenkel recommends capsules each containing myrtol, 0.15 to 0.3 gram (2½ to 5 grains); not more than 1 gram (15 grains) to be given in the course of twenty-four hours. Tar preparations, turpentine, lead acetate, and many other remedies are also recommended to be applied in the same manner.

The treatment of the cough in chronic bronchitis is an especially difficult problem; if we check the cough too much, there may be retention of secretion with all its consequences; if we permit the patient to cough too much, there may follow weakening of the bronchial coats and dilatation of the bronchial tubes. Too much coughing produces more cough, and too little coughing is followed by the same result. In addition, those remedies which reduce the cough reflex with certainty are accompanied by effects not altogether beneficial to the patient. As has been shown, diminution of expec-

toration is followed by diminution of cough. But in the dry form of chronic bronchitis there is little expectoration, and yet there is cough. When the cough is so annoying as to cause local or general symptoms, something must be done. The milder measures should first be tried: drinking of warm liquids, equal parts of Ems water and hot milk, frequently acts very well; the Species Pectorales of the National Formulary or some warm alkaline solution may be tried. In practice most patients have some especial form of tea which acts beneficially, but this fails with others, who, in their turn, are beneficially affected by a tea called by some other name but having the same ingredients.

This observation naturally led to the conclusion that excessive coughing results from some nervous mechanism which can be controlled by increased inhibition—let it be called autosuggestion. When the patient can be taught to inhibit the reflex which produces the cough, we possess an invaluable way of checking the cough and one which succeeds frequently. Most patients of average intelligence and fortitude can be taught to resist constant coughing; in some the autosuggestion must be reënforced by the administration of some drug which can be taken frequently without doing harm, in the form of a tablet, a lozenge, or a cough drop, containing horehound, althea, licorice, flaxseed, Iceland moss. In some instances hydrotherapy is valuable, the Preissnitz application around the throat or enveloping the whole chest. But there will be failures with any or all of the measures recommended, and then recourse must be had to remedies which reduce the cough reflex; the bromides and chloral are sometimes valuable, but commonly we must resort to codeine, heroin, or morphine. In a chronic disease the prevention of a drug habit is of the utmost importance; although a codeine habit does occasionally occur, it is very rare and easily relieved; therefore this drug should be administered first. If it does not give relief, heroin should be tried in small doses (0.004 to 0.008 gram; $\frac{1}{16}$ to $\frac{1}{8}$ grain); as Harnack first pointed out, it is more toxic than morphine, and heroinism (morphinism) does occur. Last of all, opium or morphine should be administered, preferably opium. There are very few cough mixtures which do not contain the one or the other.

The treatment by mineral waters is supposed to affect the expectoration, and this is probably the case when they contain alkalies or sulphur. But this indication is not the only one to be followed in sending patients to springs; frequently it is the removal of the cause which helps, or a combination of activities such as we always see following courses of treatment at springs. It is therefore difficult to be precise in indicating where the individual should be sent. Alkaline-saline and saline waters might be expected to loosen expectoration, therefore patients may be sent to Ems, Soden, Kissingen, Homburg, Carlsbad, and Marienbad for this purpose, in addi-

tion to the general ones just referred to. Sulphur waters may be expected to have more or less effect in the direction of drying up the expectoration, but they are much more valuable for general treatment. In this country we find them in the White, Red, and Salt Sulphur Springs of Virginia, Richfield, Sharon, and many others. Abroad, Harrogate, Aix-les-Bains, Cauterets, Eaux-Bonnes, and Baréges are recommended.

The character of the expectoration also largely determines the choice of climate for chronic bronchitis. To reduce expectoration dry climates should be selected; to increase it moist places should be chosen. The dry forms are best treated in Algiers, Corsica, Cannes, Nizza, Mentone abroad; in this country the coast of Florida and Southern California are of great service. In cases with moderate secretion the interior of North or South Carolina, Georgia, or Florida may be chosen. In the very moist forms the desert in Egypt or Algiers is highly recommended; in this country Montana, Wyoming, Colorado, northern New Mexico, western Kansas, southern New Mexico, and southern Arizona. Everything else being equal, we may expect complete cessation of expectoration by removal to the proper climate, therefore change from a progressive to a stationary condition and perhaps in some instances complete cure. The details for choice of climate are the same as in any other disease, and in addition to control of expectoration it should be modified by considerations in other directions, as in any other disease in a given individual.

The treatment directed with expectoration and cough as a guide not only gives relief, but prevents further development of anatomical lesions. If in addition to this we could remove the anatomical lesions produced by the chronic bronchitis and return the tissues to their normal condition we could control the whole situation. This is not possible, but something can be done. In this connection it is necessary to consider the administration of iodine and its compounds, the action of which in the human body is not thoroughly understood, but the effects of which are thoroughly appreciated by clinicians. As a result of clinical observations we have taken for granted that the administration of iodine is followed by distinct effects upon glandular structures, as well as upon some of the consequences of inflammation. The former action, I believe, has not been assailed; it is certain that in so far as the lesion of chronic bronchitis is confined to glandular hypertrophy and infiltration, the disease may be cured by the consistent administration of iodine. In some degree the changes in the bronchial wall resemble those of arteriosclerosis, as there is destruction of elastic and muscular tissue and substitution of fibrous tissue for them. We may look upon the therapeutic problem as possessing the same terms; we try to reduce connective tissue and increase elasticity and muscularity of the bronchial walls. That this follows the proper administration of iodine in arterio-

sclerosis as well as in chronic bronchitis is certain. How it occurs is not proved. Müller and Inada, working with Romberg (1904), showed that potassium iodide reduces the viscosity of the blood. Aside from the fact that this would not explain most of the changes following the administration of iodine, Determann, working with more accurate methods, failed to verify this observation. Rumpf states that iodine does not act by causing absorption, and as this view is in direct opposition to all our experience, more evidence than a mere statement should be adduced until it can be entertained. It is true there is little experimental evidence one way or the other, although Scharz (1908) has shown that by pencilling the edges of aseptic wounds with iodine very small cicatrices are left; in other words, iodine causes absorption of newly formed connective tissue. But absorption follows the use of iodine in various conditions of connective tissue formation (non-syphilitic), in brain tumors, in inflammatory results in the eyes, the ears, the joints, indeed—wherever fibrous tissue is found adventitiously. How this is done is not understood, but the fact that Huchard's views of dilatation of bloodvessels by iodides are being reëstablished, with the peculiar vascular conditions existing in newly formed connective tissue, increased blood supply might account for absorption of tissue, which impedes activity of elastic and muscular tissue in the bronchial wall merely by its presence. That the iodides act only by causing increased expectoration in chronic bronchitis is refuted by the observation that they are of great value when the expectoration is already increased. But in order to get good effects from iodine it should be given as we give it in arteriosclerosis, for a long time, from one to two years, the dosage to be controlled by the physiological effects. Any preparation of iodine may be given, and the choice frequently depends upon the so-called idiosyncrasies of the patient. Whenever it is possible the iodides should be given in chronic bronchitis, as they are followed by better results than pure iodine.

Much may be accomplished by drugs, as has been stated. Equally as much may be attained in the same direction by the proper employment of exercises and gymnastics. In order to do this we should bear in mind precisely what we wish to accomplish and then try to formulate directions which will help us to gain the desired results. For chronic bronchitis, before great structural changes have taken place these results have already been described. The formula for producing these effects is a very simple one—increase expiration. In increasing expiration the lungs and bronchi are contracted. For our present purposes circulation is changed at this time, for during expiration blood pressure is increased, and, as Kronecker and Heinricius have shown, a greater amount of blood flows to the right heart during expiration. With increased circulation stasis is removed, and with it many of the pathological conditions of chronic bronchitis. Contraction of the bronchi during expiration favors expectoration,

as accumulated secretion in glands is pressed out of the mucous membrane into the lumen of the bronchi and is followed by an increased cough reflex. Nutrition of the various coats depends largely upon contraction of the bronchi; the veins are depleted and the lymphatics are emptied. When expiration is increased, the normal effects of it are increased; moreover, certain new effects are produced in pathological states which are valuable for therapeutic purposes. There is a distinct effect tending to diminish glandular and cellular infiltration, probably also existing connective tissue. Exercise of involuntary bronchial muscle takes place with the same results that follow this procedure in the myocardium. When the organic changes existing have been diminished the possibility of regeneration of elastic tissue must be taken into consideration. This occurs much more frequently than is conceded, a fact confirmed by Jores' statement that all fibroblasts can produce elastic tissue.

How can all this be accomplished? Knopf has called attention to the fact that many people do not understand how to breathe properly or they breathe paradoxically, that is, the upper part of the thorax expands while the lower part does not, because the abdominal muscles contract during inspiration. The importance of removing this form of breathing by education immediately suggests itself for chronic bronchitis, and much stress is laid upon it by Knopf in this connection. It is very easy to teach patients to increase expiration, and the quantity of increase may be increased or diminished. The simplest form is to ask the patient to take full inspirations, which should be followed by full expirations. In doing all of these respiratory gymnastics the patient should breathe fresh air, always breathe in through the nose and breathe out through the mouth, for as the chink of the glottis is normally diminished during expiration, less resistance follows mouth than nose expiration. More effect than is produced by simple inspiration and expiration follows elevation of the arms during inspiration followed by expiration with the arms at the side. There is added flexion of the lower extremities with expiration. In order to produce still greater effects upon expiration, the lower part of the thorax should be compressed manually during each expiration in combination with one of the first movements. The latter, that is, compression of the thorax, which was first introduced by Gerhardt, is very valuable in helping expectoration. These movements should not be done more rapidly than the normal respiratory rate, 18 times per minute. Exercise of the abdominal muscles by voluntary contraction may also be tried, and in some instances it is very valuable. Passive movements by massage, electricity, or by the aid of apparatus may be valuable in individuals, as much by the mental as the physical effects which follow them.

It is impossible to discuss the theoretical and practical aspects of pneumatic apparatus within the limits of this article. Since their reintroduction about thirty-five years ago a large literature has

developed, which comprises all possible refinements of thought, reason, ingenuity, and, at times, credulity. It may be freely stated that pneumatotherapy is still used extensively; furthermore, that good results sometimes follow its application. My own experience leads me to believe that the practitioner does very well without it, an opinion which seems verified by the disappearance of pneumatic apparatus from the offices of physicians.

OPEN-AIR AND HYPEREMIC TREATMENT AS POWERFUL AIDS IN THE MANAGEMENT OF COMPLICATED SURGICAL TUBERCULOSIS IN ADULTS.[1]

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There are some localities in the body in which bone tuberculosis, though operable, cannot be cured by operation alone. Tuberculous affection of the os sacrum is one type of this class of cases; that of the pelvis, such as acetabulitis complicated with suppurating coxitis, another. The persistent sinuses following excision of the hip-joint often baffle the surgeon's skill and patience.

Cold abscess formation as a result of tuberculosis of the pelvis, especially if recurring again and again, is always a grave complication, and particularly so in adults. It is true, much can be accomplished nowadays by filling the cold abscess cavity, after evacuation of the pus through a trocar, with iodoform emulsion (best iodoform suspended in sterilized glycerin), and following this process by the faithful and persistent use of hyperemia for the diseased bone or joint. Still, there are cases that resist all our efforts in this direction. It is in these cases that additional open-air treatment may become the means of saving limb or life.

The literature in this respect, at least in so far as the treatment of older patients is concerned, is rather meagre. Halstead, of Baltimore, in 1905, read a paper at the first annual meeting of the International Association for the Study and Prevention of Tuberculosis, on the Results of the Open-air Treatment of Surgical Tuberculosis, in which he set forth the beneficial influence of fresh-air treatment in tuberculous bone affections, and gave his personal experience with the method. What has been accomplished in the young with this method is well-known. The reports issued by the

[1] Read at a meeting of the Section on Surgery and Orthopedics of the International Congress on Tuberculosis, Washington, D. C., October, 1908.

various seaside resorts abroad, as also the Sea-breeze Hospital in Coney Island, maintained by the New York Association for Improving the Condition of the Poor,[2] furnish ample proof in this direction.

The following three cases selected from a number that have come under my care within the last few years will serve to illustrate the point in question.

CASE I.—*Tuberculosis of the os sacrum, involving both ilio-sacral joints and the fifth lumbar vertebra with its articulation.*

Mr. X. Y., aged forty years, who had suffered from repeated slight attacks of hemorrhage from the lungs during the last few years, came under my care in April, 1906, on account of a cold abscess over the left iliosacral joint. The os sacrum was swollen and tender in its upper part, as was also the neighboring pelvic bone. In view of the patient's reduced general condition, more serious operative intervention could not be considered at the time. The abscess was evacuated under local ethyl-chloride-cocaine anesthesia and then filled with a 5 per cent. emulsion of sterilized iodoform-glycerin. There was little reaction. The patient was put on a nourishing diet and remained under the care of a nurse. Three weeks later, and again four weeks thereafter, the procedure had to be repeated, as the abscess had refilled. Soon afterward a discharging sinus formed. As the patient had large business interests at stake, and time was an important item, excision of the iliosacral articulation was seriously considered. However, increasing swelling and tenderness of the right (opposite) iliosacral articulation rendered operative work out of question. There could be no doubt that the entire upper part of the os sacrum was involved in the disease.

In former times, such a patient was usually doomed. Today we have learned that conservative methods of treatment may yet at times accomplish a cure in these cases. Fortunately, the patient belonged to that class that need not count the cost in trying to regain health. Accompanied by a nurse, he was sent to the mountains, where he was so fortunate as to come under the care of a most thorough and learned colleague. He was kept on his back, lived in the open day and night, was put upon a forced diet, and had general massage and proper internal medication. A typical tuberculous affection of the sheath of the left Achilles tendon, which had developed within a comparatively short time, was regularly subjected to hyperemic treatment with the elastic bandage around the thigh.

After four months he had gained almost forty pounds, and had to change his diet in order to reduce his weight. The neighborhood of the sinus was tender, as before, and the opposite sacro-iliac articulation. Now hyperemic treatment by means of suction glasses was added. Regularly every day a large oval glass globe, covering the upper part of the os sacrum and both sacro-iliac articulations, including the

[2] Medical Record, March 7, 1908.

immediate surroundings, was employed six times for five minutes, with three-minute intermissions. Half a year later the sinus had closed for the first time. It reopened and healed temporarily in the course of the following two months. But since the spring of 1907 the scar has formed permanently, proving beyond a doubt that the local tuberculous bone disease had healed. No better proof could be rendered for the deep effect of suction hyperemia.

Meanwhile pain and tenderness over the sacrovertebral joint and fifth lumbar vertebra itself had appeared. Slowly a large cold abscess developed in the right lumbar region, the side opposite to that originally evacuated. It is very probable that the hyperemic treatment caused this breaking down of the tuberculous infiltration, which, in the light of Bier's teachings, is to be looked upon as a favorable sign. Slowly the pus travelled downward along the iliopsoas muscle. When the patient came to the city for surgical treatment, in May, 1907, a typical large cold abscess could easily be made out above the upper half of Poupart's ligament. Its development could be explained in no other way than by assuming that the sacrovertebral articulation, as well as the body of the fifth vertebra, had become complicated in the disease. Typical clinical symptoms corroborated this diagnosis, which was also concurred in by a prominent orthopedic surgeon who was consulted at the request of the family.

On June 7 the abscess was tapped under local anesthesia, injury to the pelvic artery being fortunately avoided. More than a quart of thin pus was evacuated through the trocar cannula, and 100 c.c. of a 5 per cent. sterilized iodoform-glycerin emulsion injected in its place. Three weeks later the process had to be repeated; a great deal of the fluid had re-accumulated. Of course, the general treatment in all its details, including suction hyperemia, the latter now also over the lower portion of the lumbar spine, was strictly and regularly carried out, and the hypodermic application of tuberculin (Beraneck's preparation), which had been begun several months before, was continued in increasing doses. Furthermore, a leather corset supported by steel bands was procured. This was at first worn continuously, the only time of interruption being during massage and cupping. Later on, the corset was left off at night. The abdominal posture was much favored by the patient. Early in July he returned to the mountains. When seen by me there in September, the abscess had re-filled to such a small extent that aspirating, though I came prepared to do so, was not done, it being deemed wiser to trust to spontaneous absorption. At this time the bacilli in the sputum had entirely disappeared; in fact, it had been impossible by the most careful search to detect them for over a year. But the patient complained of frequent accumulation of mucus in the throat and trachea. The regular use of Kuhn's lung suction mask, which latter I had at that time imported for the

patient from Germany, was then added as another link in the chain of our conservative method of treatment in this case. As is well known, this mask was designed for the purpose of subjecting the lungs to suction hyperemia according to Bier's principles.

Faithfully the mask was used by the patient, who has always helped along energetically his attendants' efforts to effect a cure, and is impatiently waiting for the verdict "cured," so that he may return to business. The mask has been applied for one hour twice daily up to the present time. The accumulation of mucus and the slight hacking cough have disappeared. The localized tuberculosis within the sheath of the left tendo Achillis also has completely subsided.

At the time of writing these lines the patient is in excellent condition. He has returned from the mountains. If no unforeseen setback occurs, the hope may be confidently entertained that another year, carefully spent with no other aim but to get well, will suffice to restore the patient to complete health. Surely, this is a most gratifying result obtained by means of conservative treatment along modern lines in a hitherto intractable tuberculous affection.

CASE II.—*Recurrent tuberculous inflammation of the tibiotarsal joint after astragalectomy combined with extirpation of the synovial membrane. Cured by means of hyperemic treatment.*

R. E. Mc.M., male, aged sixteen years, consulted me in February, 1906, for a typical inflammation of the right tibiotarsal joint. The patient is a slim, tall, anemic young man. His father has phthisis. The *x*-rays show a typically diseased astragalus as the cause (sequestrum).

In view of the rather limited means of the family, and the excellent results obtainable in these cases by operation, conservative treatment was not favored. In February, 1906, the joint was opened according to Koenig's method. The astragalus was removed, the much diseased synovial membrane carefully extirpated, and a special incision added for drainage of the joint on the outside near the tendo Achillis. The three wounds were left wide open and the cavity was filled with iodoform gauze. The first dressing remained undisturbed for two weeks.

Early in June the patient was discharged with his wounds closed and excellent motion. Still, he was not permitted as yet to use the extremity. Equipped with crutches and the elastic bandage, which he had learned to apply during the hyperemic after-treatment, he soon left for Colorado. He improved rapidly. Next spring found him horse-back riding. In May he unfortunately severely sprained his foot. Pain and tenderness set in and several abundantly discharging sinuses resulted. I sent him a set of suction cups and a new elastic rubber bandage for hyperemic treatment of the foot without allowing him to use the latter. Full directions were given and changed from time to time according to the course of the trouble. Six months later all wounds had closed, and the pain and tenderness

subsided. He was again warned against putting any weight upon the foot for some time. He is at the present time still using the artificial hyperemia.

A recurrent trouble like this would formerly have required prompt surgical attendance; several secondary operations might have become necessary, and in case of a serious turn of the accident even amputation of the leg might have had to be resorted to. Fortunately, this patient—far removed from direct medical aid as he was; he had moved to a camp in Wyoming—had sufficient intelligence properly to carry out the treatment on basis of directions given from here. This case represents another striking illustration of what fresh air in conjunction with careful and persistent hyperemic treatment can accomplish in a comparatively short time. It also demonstrates how careful a patient with a recently healed tuberculous trouble should be to avoid traumatism to the part.

CASE III.—*Tuberculosis of the right hip-joint with multiple sinus formation. Disarticulation at the hip-joint; involvement of the pelvis. Alive after ten years of out-door life. Hyperemic suction treatment for persisting sinuses; steady improvement.*

On November 8, 1897, I performed disarticulation at the right hip for a long-standing suppurative tuberculosis of the joint at the German Hospital in F. B., a man, aged thirty-six years. He had seen many physicians and tried everything imaginable. The entire upper half of the femur was complicated in the disease and the soft parts were riddled with discharging sinuses. Total removal of the lower extremity gave the only hope of relieving his condition. This was carried out with intra-abdominal compression of the common iliac artery, through an intra-muscular incision, according to the McBurney method, which worked admirably; there was very little loss of blood.[3] Unfortunately, it was seen, after the acetabulum had been fully exposed following the removal of the extremity, that the pelvis, too, was extensively invaded by the disease. The greatly run down, anemic condition of the patient forbade immediate additional resection of the iliac bone. A few months later, when the patient had sufficiently recovered, this second operation was proposed to him, but he absolutely refused further operative treatment. He asked me what else he might do to prolong his life. I advised him to buy a place at the seashore, live there all the year around, and stay in the open air as much as possible. We had to select the seashore instead of the mountains, as the patient, being dependent on his business, wanted to go to the city regularly as long as his condition would permit. I then lost track of him, and was greatly surprised when in April, 1908, I was requested by a colleague to see this patient with him in a Long Island seashore place. I had considered him long dead and gone. Instead, I found a still very anemic man, who, up to a few days ago, had gone to the

[3] Annals of Surgery, 1898, xxviii, 131.

city regularly winter and summer attending to his business, having become quite prosperous. He wanted my advice regarding some pain he had in the region of the symphysis—a new feature in his trouble. On looking him over and comparing his present condition with that of ten years ago, I found that quite a number of sinuses had closed, while others still remained open, and extended deeply into the pelvis. He had them packed day after day with yards of narrow strips of gauze by his faithful wife, who had tenderly nursed him all these many years. His lungs were not affected. Urinary examination showed absence of albumin.

I advised that the packing be stopped and Bier's suction cups used instead, giving the necessary directions. Again I deeply regretted our present inability to produce obstructive venous hyperemia—the kind that is required for the treatment of tuberculous affections—in the bones of the pelvis and hip-joint. Today, three months later, the discharge from the sinuses has greatly decreased, his pains have disappeared, and his general health, too, is decidedly improving.

Pondering over this case, I have been much impressed by the salubrious effect of open air in what seemed to me an absolutely hopeless case.

These three patients were private patients of mine. They were able to spend more or less money in an effort to regain their health. But how about the masses, the hopelessly poor? No adult patient afflicted with chronic suppurative tuberculous bone disease, nor even one with a slightly discharging persistent sinus, will gain admission to any of our many country sanatoriums. The latter are thus far designed for internal tuberculous affections exclusively, that is, consumption—not for surgical cases. How great an amount of good could be done if provision were made by the State and by our wealthy philanthropists, who have built and are supporting these institutions, to allow also the poor sufferers from surgical tuberculosis to obtain the benefit of this all-important remedy for them: fresh air.

I would, therefore, close my remarks with the plea that, for the present, two special wards—male and female—be set aside for surgical cases in all country sanatoriums for consumptives, and that the special position of surgical assistant be created. It is self-understood that such colleague would have to be a man of hospital training and fully conversant with Bier's hyperemic treatment. This arrangement would have to be continued until the State or philanthropists had separate sanatoriums erected exclusively for the conservative treatment of adults suffering from surgical tuberculosis. I plead for separate sanatoriums, for the reason that the majority of these patients are not afflicted with complicating tuberculous affection of the lungs. If a number of private rooms were set aside in such sanatoriums for surgical tuberculosis, the income derived from this source would be a material help in defraying running expenses.

THE HEART IN PULMONARY TUBERCULOSIS.

II. THE HEART ITSELF DISEASED.

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In a former paper[1] that section of this study which deals with the heart in pulmonary tuberculosis when the heart itself is not diseased was dealt with. In this section it is purposed to discuss the heart in pulmonary tuberculosis when the heart itself is diseased. In discussing this subject an endeavor has been made to separate patients developing cardiac disease during pulmonary tuberculosis from those developing pulmonary tuberculosis during cardiac disease.

CARDIAC DISEASE DEVELOPING DURING PULMONARY TUBERCULOSIS.

Heart disease developing during pulmonary tuberculosis may be found in the order of its frequency, in the pericardium, the endocardium, or the myocardium.

Myocarditis and Changes in the Myocardium. (*A*) *Non-tuberculous.* Changes in the myocardium are not infrequent at autopsy, but difficult to diagnosticate clinically. They are very rarely tuberculous in nature and consist of fatty degeneration, fragmentation of the myocardium (v. Leyden[2]), hypertrophy of the muscle nuclei (33 times in 47 cases at the Phipps Institute[3]), brown induration, and interstitial myocarditis. In 12 of these 47 cases the heart muscle was normal, and in none was tuberculosis found. Klippel[4] has described a generalized amyotrophy with hyperexcitability of the myocardium in particular, resulting in tachycardia. The fatty degeneration, which is frequent according to De Renzi,[5] may follow this muscular atrophy (Birch-Hirschfeld[6]). Unless the pericardium also be affected, these changes often pass undetected, but when cyanosis, œdema, dyspnœa, and a frequent and irregular small pulse occur, such changes are very probable. Adynamia is not common and occurs usually only late in fibrosis. Attacks resembling pseudo-angina pectoris, Kidd[7] believes, are coincidences. The

[1] Amer. Jour. Med. Sci., 1908, cxxxvi, 819.
[2] Deut. med. Woch., 1896, xxii, 1, 19.
[3] Third Annual Report, Phila., 1907, p. 165.
[4] Des amyotrophies dans les maladies générales chroniques et de leurs relations avec lésions des nerfs périphériques, Thèse, Paris, 1889. (Quoted by Sequer, Le cœur des tuberculeux, Thèse, Paris, 1903.)
[5] Path., Sympt., u Behandl. d. Lungenschw., Wien, 1894, p. 197.
[6] Centralbl. f. allg. Path., 1891, ii, 807.
[7] Allbutt's System of Medicine, 1901, v, 214.

great increase in weight that some patients exhibit who are kept perfectly quiet must at times affect the heart, which shows unmistakable signs of weakness, due, possibly, to a fatty infiltration.

(*B*) *Tuberculous.* Tuberculosis of the myocardium is very rare, occurring about once in 1000 autopsies on tuberculous subjects (Valentin[8]). It is said to occur most frequently as miliary tuberculosis, much less often as large solitary tubercles, diffuse tuberculous infiltration with scattered caseating areas, or more rarely still as a diffuse tuberculous fibrosis without caseation. These changes seldom produce functional disturbances, occur more frequently in early life (under fifteen), and have never caused rupture or aneurysm of the heart (due possibly to the low blood pressure). The larger nodules may be single or multiple, occur more often in the ventricles (left more than right) than the auricles (right more than left), and rarely ulcerate. The disease usually extends by direct continuity from the pericardium (23 out of 30 cases, Stephani[9]) or endocardium (8 out of 20, Stephani[10]) into the myocardium, but may arise from infection from the lymph or blood stream. It has little clinical significance, and is practically never diagnosticated except post mortem.

Pericarditis. The pericardium is said to be more frequently attacked in tuberculosis than the endocardium or the myocardium. In 1780 autopsies upon tuberculous subjects, all with pulmonary involvement, tuberculous pericarditis was found, according to Norris,[11] 82 times, and if to these be added 31 instances in which no etiological factor was found, and in which he thinks tuberculosis was probably the cause, tuberculous pericarditis occurred in 6.3 per cent. Billings[12] believes that fully 50 per cent. of all cases of pericarditis are overlooked clinically, and Kidd[13] that the condition is rarely diagnosticated during life. The reports of the Phipps Institute[14] fully bear this out and emphasize the fact that the symptoms are usually very obscure and overlooked. But one case is mentioned clinically in 2999, and 16 were found in 160 autopsies. In 1300 patients at the Adirondack Cottage Sanitarium no symptoms suggesting pericardial involvement occurred except once when a temporary pericardial friction rub was noted in a far advanced case. Norris,[15] in 7646 collected cases of pulmonary tuberculosis, found 31 instances of pericarditis (0.4 per cent.). It is more frequent in childhood, and Rilliet and Barthez[16] found it in 3.2 per cent. of 312 tuberculous subjects.

[8] Contribution a l'étude de la tuberculose myocardique, Thèse, Paris, 1894.
[9] Beit. z. Klin. d. Tub., 1903, i, 387.
[10] Loc. cit.
[11] Univ. Penna. Med. Bull., 1904, xvii, 155.
[12] Jour. Amer. Med. Assoc., 1901, xxxvii, 1503.
[13] Loc. cit.
[14] Second Annual Report, Phila., 1906, p. 34; and Third Annual Report, Phila., 1907, p. 45; Third Annual Report, p. 161.
[15] Loc. cit.
[16] Handb. d. Kinderk. Translated by Krupp, Leipzig, 1844, iii, 230.

The frequent involvement of the pleura would suggest a more common occurrence clinically of pericarditis, as Thym[17] found in 53 out of 94 cases of pericarditis extension from the pleura, but the rarity with which the mediastinal glands are attacked in pulmonary tuberculosis may, in part, account for this. Infection may, of course, take place through the blood stream. Tuberculosis ranks next to rheumatism in the etiology of pericarditis.

The pericarditis may be either dry or, as is more frequent, exudative, and Thayer[18] has reported a case in which 4000 c.c. of fluid was found in the pericardium. The exudate is usually serofibrinous, and if hemorrhagic is not indicative of tuberculosis (Norris,[19] Holt[20]). Bertaux[21] calls attention to the facts that in instances in which the effusion reaches 1000 c.c. it is almost always hemorrhagic, and the real condition is often revealed first by an *x*-ray examination. Tubercle bacilli are often found with difficulty. Norris[22] found the chronic obliterative form the most common, and Osler[23] has drawn attention to the fact that a simple obliterative pericarditis, while depending upon the tuberculosis, is not necessarily tuberculous itself. Men are more frequently affected than women. A tuberculous pulmonary cavity may, in very rare instances, perforate into the pericardium, producing pneumopericarditis. The occurrence of pericarditis would naturally aggravate the symptoms of the pulmonary tuberculosis, and the prognosis is unfavorable.

Lanos[24] has described a cardiotuberculous cirrhosis, which occurs most frequently in boys under twenty years of age. The tuberculous disease starts in the mediastinal glands and spreads by continuity to the heart. Cyanosis, embryocardia, absence of precordial shock, and later, ascites, œdema, and other symptoms referable to the liver occur, which lead to a diagnosis of hepatic cirrhosis. The disease is usually fatal.

ENDOCARDITIS. Clinically endocarditis is found associated with pulmonary tuberculosis much more frequently than pericarditis, but at autopsy it is less frequent. In 8154 autopsies collected by Norris[25] valvular disease was present 293 times (3.5 per cent.). The records of 60,428 patients with pulmonary tuberculosis at the German and Swiss sanatoriums and from some private sources (Anders,[26] Norris,[27] etc.) show that valvular disease occurred in 625 (1 per cent.), and in 10,687 other records from the same sources heart disease was stated to be present in 131 (1.2 per cent.). Col-

[17] Ueber gleichzeitige Tuberkulose der Lungen und des Herzbeutels. Inaug. Dissert., Jena, 1897.

[18] Tuberculous Pericarditis. Maryland Med. Jour., 1903, xlvi, 123.

[19] Loc. cit.

[20] Diseases of Infancy and Childhood, New York, 1906, p. 619.

[21] La péricardite tuberculeuse a grand épanchement, Lyon, 1906.

[22] Loc. cit.

[23] Quoted by Norris, loc. cit.

[24] De la cirrhose cardio-tuberculeuse chez l'enfant, Thèse, Paris, 1904. (Abstract in Rev. de la Tub., 1904, i, 427.)

[25] AMER. JOUR. MED. SCI., 1904, cxxviii, 649.

[26] AMER. JOUR. MED. SCI., 1902, cxxiii, 93.

[27] Loc. cit.

lectively we have 71,115 patients with pulmonary tuberculosis, of whom 656 (and possibly 44 more) (0.9 per cent.) had some disease of the heart or of its valves. While not all, the vast majority (90 to 95 per cent.) were almost certainly endocardial lesions, and the remainder comprise so small a number as to have no practical effect upon the gross figures. No attempt, of course, has been made in these figures to separate patients with recent endocarditis, secondary to pulmonary tuberculosis, from those with endocarditis, in whom pulmonary tuberculosis has developed. Evidently here, too, many instances of the cardiac disease are overlooked clinically.

Endocarditis in pulmonary tuberculosis may be due to secondary organisms, to the tubercle bacillus, or possibly, as Teissier[28] insisted, to the tuberculous toxin. While the lymphatic tissue is frequently affected in tuberculosis and the tonsils not spared, yet it is a striking fact, long pointed out, that acute articular rheumatism or rheumatic fever, notwithstanding life in the open in all sorts of weather, is exceedingly rare. I have seen but one typical instance in over 3000 tuberculous patients. On the other hand, a point which will be referred to later, a history of rheumatism in tuberculous patients who present signs of endocarditis is very common. Anders mentions that Kidd[29] found, in 12 patients out of 27 with endocaditis, a history of rheumatism, and it was present in 4 of my 7 cases. Many tuberculous patients suffer from slight myalgia and joint pains which, in regard to etiology, are difficult to diagnosticate. Poncet[30] has repeatedly called attention to a form of tuberculous rheumatism, and, if many of these patients had this disease, then it can be stated confidently that tuberculous rheumatism is far less often accompanied by endocardial changes than rheumatic fever.

Endocarditis occurring during pulmonary tuberculosis is most often a late complication, and so passed by. I have no recollection nor any definite record of endocarditis developing in a patient with pulmonary tuberculosis. Norris,[31] from 11,655 autopsy records from all sources, found recent endocarditis noted in 151 (1.3 per cent.), and Marshall[32] states that acute endocarditis is present in 5 per cent. of all patients with pulmonary tuberculosis. It is of interest to note the frequency of endocardial changes in dogs with experimental tuberculosis. In a series of 6 dogs, inoculated intravenously and treated by zomotherapy, endocarditis was found 4 times, and in 1 of these hemorrhagic pericarditis was present (Brown[33]).

Owing to the great frequency of healed latent pulmonary tubercu-

[28] Des lésions de l'endocarde chez les tuberculeux, Paris, 1894, p. 67.
[29] St. Bartholomew's Hosp. Reports, 1887, xxiii, 239.
[30] Cong. français de chir., 1897, xi, 732; Gaz. hebdom. de méd. et de chir., 20 juillet, 1902, p. 673.
[31] Loc. cit.
[32] Johns Hopkins Hosp. Bull., 1905, xvi, 303.
[33] Amer. Jour. Med. Sci., 1903, cxxv, 1071.

losis among the general population, Marshall[34] thinks the two diseases are often thus associated, although the pulmonary tuberculosis is of no clinical significance and the association of no importance.

Tuberculous Endocarditis. True tuberculous endocarditis is very rare, and many instances reported as such, as Marshall[35] has pointed out, cannot be accepted. The endocardium reacts macroscopically alike to all organisms and the vegetations present no distinguishing features (Braillon[36]). Tubercle bacilli are not infrequently in the blood in tuberculosis, as Jousset,[37] Luedke,[38] Liebermeister,[39] and others have demonstrated. That these circulating tubercle bacilli should lodge on the valves affected by a preëxisting endocarditis is not improbable, and besides the presence of tubercle bacilli, true tuberculous changes must be present in the vegetations, or if only miliary tuberculosis occur, they must lie, as Benda[40] has shown, above the elastic membrane to justify a diagnosis of tuberculous endocarditis. When this test is applied, fewer instances of tuberculous endocarditis are found.

Tuberculous endocarditis may be divided into (1) miliary, (2) true tuberculous endocarditis, (3) endocarditis due to extension from the myocardium, and (4) endocarditis due to extension from an organizing tuberculous thrombus (Marshall[41]). The miliary type is the most frequent, and occurs generally in connection with miliary tuberculosis. Braillon[42] believes that the course of tuberculous endocarditis can be divided into two periods, the first, virulent, resembling septicemia, during which tubercle bacilli are contained in the blood, which may facilitate diagnosis, and second, a period during which the lesion tends toward healing and sclerosis. Tuberculous endocarditis is, however, so rare as to have no clinical significance, and has seldom been diagnosticated ante mortem, as it may occur without producing murmurs, and the fever accompanying it is attributed to the pulmonary tuberculosis. It may be nodular, caseating, or vegetating, and has been produced experimentally in rabbits (Michaelis and Blum[43]).

Endocarditis due to secondary organisms may occur, or recur, from an old lesion, is usually acute, vegetating, rarely ulcerative, and resembles closely an ordinary endocarditis. Marshall,[44] who found acute endocarditis in 5 per cent. of patients with pulmonary tuberculosis, would place here the majority of these cases.

Mitral Stenosis. Teissier[45] has described as very common (40 per cent.) a sclerotic valvular endocarditis, due to a slowly acting

[34] Loc. cit.
[35] Loc. cit.
[36] Rev. de la tub., 1904, i, 254.
[37] Cong. Int. de la Tub., 1905, i, 428.
[38] Wien. klin. Woch., 1906, xix, 949.
[39] Int. Centralbl. f. d. gesamte Tub., Forschung, 1907, ii, 36.
[40] Lubarsch and Ostertag, Ergeb. d. allg. Path., etc., 1898, v, 455. Berl. klin. Woch. 1899, pp. 566, 596, 646.
[41] Loc. cit.
[42] Loc. cit.
[43] Deut. med. Woch., 1898, xxiv, 550.
[44] Loc. cit.
[45] Des lésions de l'endocarde chez les tuberculeux, Paris, 1894, xxx, 59.

tuberculous poison, which some believe is comparable to the ether extract, obtained by Auclair[46] from the tubercle bacillus. The valve usually affected is the mitral, and the lesion produced stenosis, which, as I shall mention later, has some retarding effect upon the pulmonary lesion. It is said to occur rarely as the primary disease, and to be more inhibitory to pulmonary tuberculosis than any other form of cardiac disease. Warthin[47] accepts this form of endocarditis, but does not accept toxemia as the cause. Chartier,[48] however, holds that the sclerosis is an index of resistance to the tuberculosis, and Potain[49] states that it never occurs in acute but frequently in fibroid forms of pulmonary tuberculosis. Pure mitral stenosis, according to Malmonte,[50] is more often the manifestation than the cause of an attenuated bacillosis, which expresses itself in the form of lupus, arthritis, or various hereditary manifestations (chlorosis, scrofulosis, etc.).

Thrombi. Thrombi in the right ventricle are rarely, if ever, diagnosticated ante mortem. They rarely occur in acute cases, but are usually found very late in chronic cases with marked cachexia, and are due to changes in the endothelium, to stasis provoked by the pulmonary lesion and hence practically always in the right ventricle, and to changes occurring in the blood, such as a lessened amount of blood, an increased amount of fibrin, fewer erythrocytes, less hemoglobin, leukocytosis, lessened alkalinity, and the presence of tubercle bacilli (Barie[51]). Warthin[52] found thrombi in 10 per cent. of his tuberculous patients. A number of instances have been reported in which the thrombus had organized, and tubercle bacilli and caseation were found in the thrombus, in some cases extending into the endocardium. Barie[53] says the symptoms are obscure, but a sudden onset of dyspnœa with crises of suffocation and a tumultuous heart action, without signs in the pleura or lungs to explain it, is very suggestive. Syncope or pulmonary apoplexy (embolism) may occur. Slighter forms of pulmonary embolism are often diagnosticated as infarcts, or overlooked entirely, especially in the moribund (Kidd[54]).

PULMONARY TUBERCULOSIS DEVELOPING DURING CARDIAC DISEASE.

The great frequency of pulmonary tuberculosis makes it probable that, other things being equal, a large number of patients with heart disease should also have pulmonary tuberculosis. The greatest age incidence in pulmonary tuberculosis is, roughly speaking, between the twentieth and fortieth years, while for heart disease the

[46] Médecine moderne, 1901, xii, 118.
[47] Buck's Ref. Handb. of Med. Sci., iv, 602.
[48] Rev. de la tub., 1904, i, 41.
[49] Quoted by Norris, loc. cit.
[50] Tuberculose pulmonaire et rétrécissement mitral pur., Lyon, 1905.
[51] Jour. d. Praticiens, 1904, xviii, 257.
[52] Med. News, 1899, lxxiv, 453.
[53] Loc. cit.
[54] Loc. cit.

most frequent period is either earlier or later. So it is seen that the age incidences of the two diseases do not exactly coincide.

The extreme rarity of primary tuberculous carditis renders discussion of this condition of little practical value and makes the occurrence of secondary pulmonary tuberculosis purely problematical. Primary non-tuberculous pericarditis is often latent and may be of short duration; the occurrence of secondary pulmonary tuberculosis has received and deserves scant consideration. Two patients, both of whom have made good recoveries, gave a history of an attack of "pericarditis" before entrance to the Adirondack Cottage Sanitarium.

ENDOCARDITIS. The main interest in pulmonary tuberculosis secondary to cardiac disease is in connection with endocarditis. In the vast majority of all cases in which these two diseases are associated, I believe that the cardiac disease is clinically, at least, the primary. This statement may be contrary to that held by some observers, but in over 3000 patients, who have been more or less closely followed for a number of years, I have yet to see an instance in which undoubted valvular disease developed in a patient with pulmonary tuberculosis. The great frequency of pulmonary tuberculosis, and its early incidence, makes it more probable that it is usually pathologically the primary disease, but certainly a smaller percentage of patients with cardiac disease present signs of pulmonary tuberculosis than are found in the general population Consequently, when Rokitansky[55] says that persons laboring under enlargement of the heart (dilatation, hypertrophy, and their complications), whether primary or superinduced by mechanical obstruction at its orifices, do not contract tuberculosis, he may be right, but if so, a number of these patients must have contracted it before the development of cardiac disease, and further, it does seem as if in some it retarded the development of the pulmonary tuberculosis. The cause of this is difficult to determine, but it is a well-known fact that when pulmonary tuberculosis becomes complicated with tuberculous disease elsewhere, the primary lung lesion often retrogrades or remains stationary, while the complication advances, and on the other hand, when the complication becomes less active, the primary disease may light up. Otto[56] believes that the cardiac disease prevents the spread of the pulmonary tuberculosis. It cannot be denied that implantation may occur, as Otto[57] holds, during a period when compensation fails and when poor nourishment of the heart does not enable it to keep up its work.

The frequency of the association of endocarditis and pulmonary tuberculosis has been previously dwelt upon, but it is interesting to note in passing that the percentage of endocarditis occurring in

[55] Manual of Pathological Anatomy, Swaine's translation for the Sydenham Society, 1854, i, 316.

[56] Virchow's Archiv., 1896, cxliv, 159.

[57] Loc. cit.

cases of pulmonary tuberculosis has been stated as from 0.1 to 6.4 per cent. Anders,[58] after excluding 29,000 cases of pulmonary tuberculosis out of 30,000, arrives at the conclusion that the approximately correct percentage is 3.13, but from the 71,000 cases previously mentioned, including Ander's,[59] the percentage was 0.9. From this it appears to me that about 1 per cent. of patients with pulmonary tuberculosis present signs of valvular disease. Of course, such statistics are open to criticism, and in some instances symptoms referable to the heart are unquestionably ascribed to the lungs, and the heart consequently examined in a cursory manner. But it must not be forgotten that the vast majority of these patients were in very early stages. At the Phipps Institute[60] 9 per cent. of 1009 patients were found clinically to have had valvular lesions, but no mention is made, so far as I could find, of the percentage of these that showed valvular disease at autopsy. Meisenberg,[61] who has made a careful study of the records of 43,365 patients at Leipzig, found 1.75 per cent. with cardiac disease and 13 per cent. with pulmonary tuberculosis; 7 per cent. of the patients with cardiac disease had pulmonary tuberculosis and 1.14 per cent. with pulmonary tuberculosis had heart failure. Von Kryger,[62] in 1100 autopsies on tuberculous subjects, found only 10 (0.9 per cent.) with cardiac disease, but in 59 autopsies on patients dead of marked cardiac disease, all had healed or arrested pulmonary tuberculosis. Frommolt,[63] in 7870 autopsies at Dresden, found valvular disease in 277, and of these, only 8 per cent. had pulmonary tuberculosis. Birch-Hirschfeld,[64] in 4359 routine autopsies, found pulmonary tuberculosis in 20.8 per cent., while in 107 autopsies on subjects with cardiac disease, pulmonary tuberculosis was found in 4.6 per cent. Norris,[65] who has collected the data of 8154 autopsies upon tuberculous subjects, found valvular disease present in 3.65, and in 388 autopsies on subjects with cardiac disease, pulmonary tuberculosis was noted in 6.7 per cent. It is difficult to explain the great differences among these figures, but if we recall the indifference with which tuberculosis has been treated at many general hospitals, and the reluctance some pathologists have for performing autopsies in apparently uncomplicated cases of pulmonary tuberculosis, the larger percentage of heart disease found by some pathologists may be readily explained.

Meisenberg[66] from his studies concluded that, given either disease, the other is less frequent than usual, and Anders believes that Graham's[67] figures tend to uphold this contention.

[58] Loc. cit.
[59] Loc. cit.
[60] Second Annual Report, Phila., 1906, p. 239.
[61] Ztschr. f. Tub. u. Heilst., 1902, iii, 329.
[62] Inaug. Dissert., Munich, 1889 (1887). Quoted by Norris, loc. cit
[63] Arch. f. Heilk., 1875, xvi, 238.
[64] Loc. cit.
[65] Loc. cit.
[66] Loc. cit.
[67] Montreal Med. Jour., 1896, xxv, 101.

The question of sex in regard to this subject has been studied by Otto,[68] who believes that pulmonary tuberculosis is more frequently associated with cardiac disease in women than in men. Five of my 7 cases were in men.

The left side of the heart is affected in the vast majority of cases; in fact, the right side of the heart is rarely attacked. Congenital pulmonary stenosis, a very infrequent disease, forms a large percentage, 30 to 40 per cent. at least, of all the lesions found upon the right side of the heart.

Some difference of opinion formerly existed regarding the relative frequency of involvement of the mitral and the aortic valves, but now it is well recognized that disease of the mitral valve is more frequent than that of the aortic (5 to 2 in my cases), and both are more often affected singly than together. The more frequent occurrence of mitral disease during the greatest age incidence of pulmonary tuberculosis would readily explain this. In 265 autopsies Norris[69] found the mitral valve affected in 183, the aortic in 126, the tricuspid in 23, and the pulmonary in 13. Pure mitral insufficiency, according to Meisenberg,[70] is the most frequent lesion associated with pulmonary tuberculosis; more rarely aortic insufficiency occurs, and still more rarely mitral stenosis, with or without simultaneous insufficiency, and most rarely a simultaneous affection of several valves.

Mitral Insufficiency. The coincidence of the age incidence of pulmonary tuberculosis and of this lesion has already been mentioned. Some hold that for anatomical and physiological reasons this lesion more than any other is likely to occur with pulmonary tuberculosis, while it does not apparently predispose to pulmonary tuberculosis.

However this may be, mitral insufficiency is certainly the most frequent cardiac lesion in pulmonary tuberculosis. In 36 instances of "organic murmurs" at the Phipps Institute,[71] pure mitral insufficiency occurred 24 times, and was present in connection with other murmurs 7 times. In 77 collected instances of valvular disease in connection with pulmonary tuberculosis, I found pure mitral insufficiency in 44, and in 4 others it was associated with other lesions. In Norris'[72] report on 293 valvular lesions, mitral insufficiency was found 84 times alone, and in conjunction with other lesions 13 times.

It seems, if well compensated, to exert little influence upon the course or the symptoms of the pulmonary tuberculosis, but Anders[73] has noted more and more profuse hemoptysis, increased dyspnœa of a cardiac type, and lower temperature. I have not noted in my few cases such symptoms. When compensation is broken, however, the pulmonary tuberculosis may quickly advance. In the Leipzig

[68] Loc. cit.
[69] Loc. cit.
[70] Loc. cit.
[71] Third Annual Report, Phila., 1907, p. 40.
[72] Loc. cit.
[73] Loc. cit.

figures, pulmonary tuberculosis occurred in 16 per cent. of all cases of mitral insufficiency. Among the 7 cases of valvular disease occurring in the last 1300 patients at the Adirondack Cottage Sanitarium, mitral insufficiency was present 4 times (3 in males). In 3 of these a history of inflammatory rheumatism long antedating the pulmonary tuberculosis was obtained, and in the other patient a history of indefinite "rheumatic" pain was noted for two years or longer, while the first symptom referable to the pulmonary tuberculosis was noted over four years previously. The indefinite history in regard to the rheumatism makes this case of little value in regard to this point. Of the 4 patients, 2 are dead, 1 is doing very well, while the other has such advanced disease that her activities are necessarily limited.

Mitral Stenosis. Stenosis of the mitral valve, far less frequent than insufficiency, has long been looked upon as antagonistic to pulmonary tuberculosis, that is, that pulmonary tuberculosis is more rarely associated with mitral stenosis than with other forms of heart disease. If any disease that produces cyanosis affords protection against pulmonary tuberculosis, as Rokitansky[74] believes, then pulmonary tuberculosis should rarely be associated with mitral stenosis. This increased venosity of the blood has been shown by Anders[75] to occur in pulmonary stenosis, which is generally complicated by pulmonary tuberculosis, and pulmonary tuberculosis is not most frequently associated with aortic regurgitation, as James[76] stated. Meisenberg[77] has attributed the relative infrequency of pulmonary tuberculosis in mitral stenosis to increased blood pressure, reduced blood stream, slowing of the blood stream, and chemical changes in the blood. Otto[78] and Peter[79] think that the congestion of the bases throws increased work upon the apices, and so better aëration prevents against tuberculosis. More recently Graham[80] has collected some interesting explanations. Congestion of the lung produces dilatation and tortuosity of the capillaries which project into the alveoli. There is also cell proliferation, with slight hypertrophy of fibrous and muscular tissue. Implantation and proliferaof tubercle bacilli are retarded because the increased flow of serum exerts an increased germicidal effect, the cells are better nourished, and the bronchioles are better flushed out; the increase of muscular tissue may better aid in forcing out foreign matter; the fibrous hyperplasia may prevent the growth of or wall off already existing foci; and the chronic congestion may exert some inhibition, since Hess[81] has shown that the resistance of the lung is proportional to the amount of elastic tissue present, and Pierce[82] that in congestion

[74] Loc. cit.
[75] Loc. cit.
[76] Quoted by De Renzi, Path., Sympt., u. Behandl. d. Lungenschw., Wien., 1894.
[77] Loc. cit.
[78] Loc. cit.
[79] Gaz. des hôp. de Paris, 1875, xlviii, 769, 785
[80] Loc. cit.
[81] Beitr. z. Klin. d. Tub., 1904, ii, 103.
[82] Proc. Path. Soc. Phila., 1901, N. S., iv, 215.

more elastic tissue is found. Anders[83] does not believe that the passive congestion is the true inhibiting factor, as hemoptysis relieves the symptoms, and Malmonte[84] attributes little, but Teissier[85] and Tileston much, to congestion. Tileston,[86] in a recent very suggestive article, reviews the autopsy records of the Massachusetts General Hospital, the Boston City Hospital, and the Long Island Hospital of Boston, which bear upon this point. In 128 cases of mitral stenosis he found a much lower percentage of pulmonary tuberculosis than in the material from which they were drawn. Patients with a high degree of stenosis had much less often pulmonary tuberculosis and in no instance active tuberculosis. They seemed also less liable to contract pulmonary tuberculosis, but if they did so, the disease ran a mild course with a strong tendency to cure. Tileston[87] has excluded the "rheumatic diathesis" and cardiac hypertrophy, and does not think that increased arterial pressure in the lungs is the factor of importance. The relative immunity, which he believes exists in these patients, he attributes to the passive hyperemia of the lungs consequent upon the mitral lesion. The cardiac hypertrophy is, according to Abrams,[88] the essential factor. I know of no observations upon the opsonic index in these conditions. Norris'[89] figures do not indicate that mitral stenosis is overlooked clinically in connection with pulmonary tuberculosis more frequently than other forms of valvular disease, and it is difficult to accept Teissier's[90] figures, who finds some sclerosis of the mitral valve in 40 per cent. of all patients with pulmonary tuberculosis, and attributes it to a sclerogenic poison of the tubercle bacilli. In 54 instances of cardiac disease Potain found pulmonary tuberculosis in 9 (16 per cent.), and Meisenberg[91] found it in 2.6 per cent. of his Leipzig cases. Patients with mitral stenosis often do remarkably well, better even than those with mitral insufficiency who often upset many prognostic data.

Three patients, two men and one woman, have been sent to the Adirondack Cottage Sanitarium with a diagnosis of mitral stenosis. In one the diagnosis had been made years previously, and the patient with advanced laryngeal and pulmonary tuberculosis did poorly, returned home, and died. One of the other two patients has done very well. The third is still at the Sanitarium. In none of them was I certain of any cardiac lesion.

A point of interest connected especially with this type of cardiac disease is the treatment of tuberculosis by passive congestion, first suggested by Bier. Its great success in the treatment of various tuberculous processes has led many to attempt to apply such pro-

[83] Loc. cit.
[84] Loc. cit.
[85] Des lésions de l'endocarde chez les tuberculeux, Paris, 1894.
[86] Tileston, Jour. Amer. Med. Assoc., 1908, l, 1179.
[87] Loc. cit.
[88] Quoted by Anders, loc. cit.
[89] Loc. cit.
[90] Loc. cit.
[91] Loc. cit.

cedures to the lungs. A prone position has long been advocated for various reasons, but Jacoby[92] has attempted, by elevating the hips and legs above the thorax, to accentuate the hyperemia produced by the prone position. More recently, Kuhn[93] has suggested his mask covering both mouth and nose, which admits air slowly through valves which may be regulated, but permits free egress of all air. This has been widely used, but lacks as yet full confirmation.

Aortic Insufficiency. This lesion is much rarer than many older authorities stated, and occurs, according to Meisenberg[94] in 5.4 per cent., according to Norris in 3.6 per cent., of all patients with cardiac lesions in pulmonary tuberculosis, and in 0.09 per cent. of all patients with pulmonary tuberculosis. Neither inhibiting nor predisposing influences have been attributed to it. In the 1300 cases this lesion has occurred twice; one patient is at present in the Sanitarium and the other returned home with an arrested process. De Renzi[95] and others believe, however, that this type of valvular disease is the most frequent in pulmonary tuberculosis, but Anders[96] combats this view and holds that when pulmonary tuberculosis is associated with it, the lung disorder aggravates the cardiac disease.

Aortic Stenosis. This lesion, as would be supposed, is apparently much less frequent than insufficiency, judging from both the clinical and autopsy records. Its frequency clinically may be judged from the fact that Meisenberg[97] saw it twice in 4649 cases of pulmonary tuberculosis, and von Ruck[98] once in 3000. I have seen no instance of it.

Pulmonary Stenosis. Pulmonary stenosis is rare, occurring, according to Norris,[99] in 0.12 per cent. of patients with pulmonary tuberculosis, and in 4.8 per cent. of patients with pulmonary tuberculosis and cardiac disease. Of 459 cases, 37 per cent. died of pulmonary tuberculosis. On the other hand, Anders,[100] who has added 36 cases from the literature to the 24 collected by Lebert,[101] thinks pulmonary tuberculosis always follows congenital pulmonary stenosis, and there is little doubt that the longer a patient lives with pulmonary stenosis the more likely he is to die of pulmonary tuberculosis. Lebert[102] believes that one-sixth of the subjects, if they live to reach their twentieth year, die of pulmonary tuberculosis, while Stolker[103] holds one-seventh. Vierordt[104] held that tuberculosis was no more frequent in patients with pulmonary stenosis than in those with any other congenital heart disease, due to their lack of vitality and resistance, and Otto[105] states that tuberculosis is the rule in all

[92] Münch. med. Woch., 1899, xlvi, 628, 659.
[93] Münch. med. Woch., 1907, liv, 782.
[94] Loc. cit.
[95] Path., Sympt., u. Behandl. d. Lungenschw., Wien, 1894, p. 195.
[96] Loc. cit.
[97] Loc. cit.
[98] Personal communication to Norris, loc. cit.
[99] Loc. cit.
[100] Loc. cit.
[101] Berl. klin. Woch., 1867, p. 233.
[102] Loc. cit.
[103] Quoted by Norris, loc. cit.
[104] Nothnagel, Spec. Path. u. Therap., 1901, xv, 100.
[105] Loc. cit.

lesions of the right side. According to Lebert,[106] while 30 per cent. of these patients die of tuberculosis, it is not necessarily of the lungs, and when in the lungs the cause is not to be attributed to anemia, but to the unequal and abnormal character of the circulation. The lungs receive their blood supply, as Norris[107] points out, from the bronchial arteries. The pulmonary tuberculosis is usually miliary in type (Fox[108]).

Frommolt[109] states that disease of several valves is not uncommon in connection with this lesion, but otherwise seldom occurs. Meisenberg[110] found only 20 in 182 instances of cardiac disease in pulmonary tuberculosis, in which the aortic and mitral valves were simultaneously affected. Jaccoud has observed many instances of tricuspid insufficiency with venous pulse, and Osler[111] mentions the frequency with which the venous pulse is observed in pulmonary tuberculosis. Other observers have made little or no note of it. Tricuspid insufficiency may be a source of great danger in benign chronic cases with sclerosis.

To summarize, it may be said that pulmonary stenosis is always the primary disease, mitral insufficiency and aortic insufficiency usually the primary. The most frequent cardiac lesion secondary to pulmonary tuberculosis in patients up and about is mitral stenosis. Pulmonary tuberculosis occurs more frequently in patients with pulmonary stenosis than in any other form of cardiac disease. Mitral insufficiency is associated with pulmonary tuberculosis more often than any other form of valvular disease, but aortic insufficiency is not very much less frequent. Aortic stenosis is very rarely associated with pulmonary tuberculosis, and pulmonary stenosis is infrequent in pulmonary tuberculosis. Involvement of several valves simultaneously is rare in these cases.

Diagnosis. The heart plays an important part in the diagnosis of pulmonary tuberculosis, and in many instances it is impossible to arrive at any definite conclusion. Marshall[112] reports several instances at the Johns Hopkins Hospital in which cardiac disease was diagnosticated as pulmonary tuberculosis and the error discovered at autopsy. Three instances have come under my notice in which a similar diagnosis was made and in which, after a most careful study, pulmonary tuberculosis could be excluded.

Treatment. The treatment of the various forms of valvular heart disease, occurring in connection with pulmonary tuberculosis, differs in no way from that ordinarily employed in cardiac disease. Moderate elevations, not over 2000 feet, are said by Anders[113] to be the best for patients with valvular disease, but if well compensated

[106] Loc. cit.
[107] Loc. cit.
[108] Diseases of the Lungs and Pleura, London, 1891, p. 547.
[109] Loc. cit.
[110] Loc. cit.
[111] Principles and Practice of Medicine, 1905, p. 333.
[112] Loc. cit.
[113] Loc. cit.

they often do well at greater elevations. Patients should be fully warned against overexertion, for they have even less reserve force than the ordinary patients with only cardiac disease, and slight efforts may produce signs of non-compensation in both systems, rendering them much more susceptible to secondary infection (Bohland[114]). The treatment of the rapid heart action and cardiac weakness in pulmonary tuberculosis differs but slightly from that ordinarily employed. Digitalis and strophanthus are of little avail. Arsenic, strychnine (in large doses), an ice-bag over the precordium, carbonic acid baths, rubs, absolute rest at first, and carefully regulated exercises relegated chiefly to those muscles which throw little or no stress upon the muscles of the thorax, are all of value. Nitroglycerin may give marked relief. Excitement of any sort should be avoided, and it is wise to refrain from the use of alcohol and tobacco and to use coffee in moderation, if at all. Burton-Fanning[115] has pointed out the value of restriction of food, and in some cases it is wise to omit milk for a time. Flatulency must be rigorously combated and the diet should be rich in easily digested proteids. Palpitation of the heart should be treated by rest and the ice-bag, but bromides and valerian may be required.

BACILLUS COLI COMMUNIS: THE CAUSE OF AN INFECTION CLINICALLY IDENTICAL WITH TYPHOID FEVER.

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Cases of infection clinically identical with typhoid fever have been caused by Bacillus fæcalis alcaligenes, the paratyphoid and paracolon bacilli (Bacillus paratyphosus, types A and B), Bacillus enteritidis, and Bacillus psittacosis. The close biological relationship of these members of the typhoid-colon group (the Typhaceæ, as Loeffler has designated them recently) and the fact that the paracolons are more nearly related to Bacillus coli than to Bacillus typhosus naturally lead to the suspicion that some strains of Bacillus coli also might be capable of causing the typhoid symptom complex. Though there is increasing evidence of variability, and even mutation, of

[114] Handb. d. chron. Lungenschw., 1904, p. 694.
[115] Open-air Treatment of Pulmonary Tuberculosis, Chicago, 1905, p. 103.

type among the Typhaceæ, a discussion of this phase of the typhoid problem does not come properly within the scope of our paper.

Our attention was first directed to the question of systemic infection by the colon bacillus pursuing a typhoid-like course in 1903, when Buxton and one of us recovered colon bacilli from the blood of two cases in Bellevue Hospital which were considered clinically as examples of mild typhoid fever. These cases were reported briefly on a former occasion:

CASE I.—A. F., butcher's helper, entered Bellevue Hospital in the service of Dr. Thompson, August 15, 1903, the eighth day of his illness. He had been swimming daily in the East River. For a week he had suffered from general weakness, frontal headache, pain in the stomach and legs, bad taste in the mouth, and loss of appetite, but in spite of these symptoms had continued at work washing beef tongues in a damp cellar. There had been no vomiting or diarrhœa.

On admission the patient's tongue was coated and tremulous. The spleen was not palpable. About two dozen rose spots were found upon the chest and abdomen. The general appearance of the patient suggested mild typhoid fever. Fresh rose spots appeared on August 17, but by the 20th all the spots had disappeared. The temperature on admission was 100.4° F; pulse, 96. Thereafter the temperature gradually fell and reached normal on the twelfth day of the disease. The patient's serum was tested against the typhoid bacillus on the tenth and twelfth days, microscopically in dilution of 1 to 100, both times with negative results after two hours. Unfortunately the reaction of the serum against the colon bacillus was not tested. On the tenth day of the disease 10 c.c. of blood, taken from a vein at the elbow, contained a colon bacillus in pure culture. August 30, after twelve days of normal temperature, a relapse occurred. During the relapse the temperature rose to a maximum of 101° F. The duration of the relapse is unknown, since on September 14, thirty-eight days after admission, the patient insisted on leaving the hospital with a pulse of 120 and a temperature of 99.5° F.

CASE II.—B. McM. Occupation, housework. Entered Bellevue Hospital August 14, 1903, in the service of Dr. Thompson, complaining of severe general headache, dry cough, pain in the back and sides, loss of appetite, and constipation. She had a temperature of 101.4° F. and a pulse of 100. A clinical diagnosis of mild typhoid fever was made. On the sixth day of illness her serum, in dilution of 1 to 100, was negative microscopically to the typhoid bacillus after two hours. Two days later her blood was found to contain a colon bacillus in pure culture. But the following day her serum gave a positive Widal reaction at 1 to 100. The reaction against Bacillus coli was not tested.

According to present standards, the data concerning Case II

are too meager to permit our drawing any conclusion regarding it. We are unable to determine whether the case should be considered as one of primary systemic infection with a colon bacillus, exhibiting a group reaction against the typhoid bacillus, or whether to look upon the presence of Bacillus coli in the blood as a secondary invasion. The former seems the more likely when one considers the small number of patients whose blood has contained Bacillus coli communis during life, and the very infrequent association of Bacillus typhosus and Bacillus coli in the blood of typhoid cases.

Case I, on the other hand, meets the requirements more fully; that is, (1) it was clinically mild typhoid fever, (2) Bacillus coli communis was obtained in pure culture from the blood, and (3) the serum reaction was negative to the typhoid bacillus as late as the last day of the fever in the primary attack. It would have added to the completeness of the data if the patient's serum had been tested against several strains of Bacillus coli, but this was not done. With these cases in our possession, a search of the literature for similar ones was undertaken.

In 1902 Fox recovered a colon bacillus from the blood of a case which resembled typhoid fever clinically. The serum of the patient gave a positive Widal reaction on the thirty-second day of the disease, though it had previously been negative. Therefore, as in Case II above, the true nature of the infection was doubtful.

In the same year Burch wrote, under the title of "Colon Bacillus Infection," that for ten years he had seen in his practice cases of fever, lasting from seven to ten days, which were ushered in by malaise, a gradual rise of temperature to a maximum of 103° to 104° F. by the third or fourth day, diarrhœa, or, less frequently, constipation. The tongue was dry and often coated. Sometimes there was mild delirium; there was always more or less headache. Tympanites occurred in some cases. The whole clinical picture was that of typhoid fever. The leukocytes were diminished in all cases. The urine was always acid and many times swarmed with motile organisms resembling Bacillus typhosus. The diazo reaction was, as a rule, well marked. The Widal reaction, made daily (dilution approximately 1 to 5), was absent in every case throughout its course, but the serum reacted promptly to the organism obtained under aseptic conditions from the bladders of the patients. This organism responded to all the cultural tests for Bacillus coli communis. This remarkable series of cases was observed in Baldwinsville, New York. While the nature of the infection cannot be accepted as proved, the cases appear to have been what the author considered them, examples of colon bacillus infection pursuing the course of typhoid fever.

In 1903 three papers appeared upon this subject. Evans and Sailer reported the case of a negro who, on February 12, suddenly became weak and dizzy. Next day he had diarrhœa, loss of appe-

tite, and gastric disturbances. The spleen was palpable. Rose spots were present. The disease ran the course of typical typhoid fever. The temperature became normal on March 6. A relapse occurred which lasted to the 16th. The leukocytes on February 15 numbered 5900; February 17, 10,480; March 9, 7000. The diazo reaction was positive in the first part of the illness. The serum tests upon which the diagnosis rests must be given in detail: Against Bacillus typhosus the reaction was positive at 1 to 10 on five occasions, while it was negative at higher dilutions. Against Bacillus paratyphosus (four strains), the reaction was incomplete at 1 to 10 on the twentieth day of the disease, but it was positive at 1 to 10 on the forty-third day of the disease. Against Bacillus coli communis (Pasteur Institute culture), the reaction was complete in forty-five minutes at 1 to 100 on the twentieth day. On the forty-third day, the reaction was complete at 1 to 150 in one hour, partial at 1 to 200 and 250 in one and a half hours (no increase later), and negative at 1 to 300 and 1 to 400 after twenty-four hours. The case, therefore, appears to have been caused by a colon bacillus.

A. Jacoby, of New Orleans, reported a case which he considered especially interesting because it resembled typhoid fever so closely: A laborer, aged fifty years, was working on a railroad in Texas when he was taken ill. He had drunk any water he could get. There had been no diarrhœa. The temperature on admission to hospital was 101° F.; pulse rapid. The abdomen was distended. The spleen was much enlarged. Rose spots were present. No malarial organisms found. The Widal reaction was negative four times (dilutions and days not stated). Rose spots and the urine were examined for Bacillus typhosus with negative results. Bacillus coli communis was present in the urine in large numbers. After admission the temperature did not rise above 102° F., and in a week began to decline. On the sixteenth day, however, double lobar pneumonia developed, the temperature rose suddenly to 104° F., and death followed. At autopsy, the small intestine was congested throughout its extent, but Peyer's patches were not involved.

Bauermeister reported the case of a man, aged thirty-five years, from whose blood he obtained Bacillus coli communis in pure culture on several occasions; he regarded the case clinically as typhoid-like in its course. Similar attacks recurred over a period of one and one-half years, when one of the patient's testicles had to be amputated because of an abscess which contained Bacillus typhosus. Bauermeister suggests that the original colon bacillus had changed its biological characters probably as the result of long residence in the body. This case seems to us to belong with the colon bacillus septicemias rather than in the typhoid group which we are discussing.

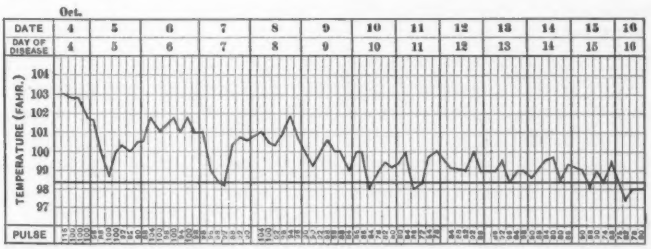
In 1904 Widal and Lemière reported two cases, which they state resembled typhoid fever, and from whose blood they recovered colon

bacilli. As their second case showed multiple abscesses of the kidney at autopsy, it cannot be included in the present series. Their third case occurred in a man, aged thirty-five years, who was taken ill suddenly five days before admission to the hospital with violent abdominal pain, repeated chills, and vomiting. He had almost constant headache and complete insomnia after the onset. There was no epistaxis or diarrhœa. The temperature on admission was 103.5° F.; pulse 90. There was marked abdominal distention. No rose spots. Spleen voluminous. Violent intestinal peristalsis. Heart, lungs, and liver normal. The abdominal pain and tenderness disappeared. Diazo reaction negative. Serum, negative to the typhoid bacillus. The patient's illness lasted from December 12 to December 21. He was discharged from the hospital December 24. On December 18 a colon bacillus was obtained in pure culture from 3 c.c. of blood. December 24, the patient's serum agglutinated his own bacillus at 1 to 30.

These are the only cases we have been able to find, reported as cases of systemic infection by the colon bacillus, pursuing the course of typhoid fever, which will bear analysis.

In the fall of 1905 we were fortunate enough to have the privilege of studying the following case:

Case III.—G. K., aged twenty-nine years, entered Bellevue Hospital October 4, in the service of the late Dr. Loomis. He had been in Central America for a year and a half, where he contracted malaria and yellow fevers. Early in July he spent eight days in Jamaica, *en route* to New York, where he has remained since August 24. During this time he has been well. The present illness dates from September 30, when he felt feverish. He waked



Temperature chart of Case III (G. K.).

next morning with a headache, mainly frontal and occipital; pain in the muscles of the neck on moving the head, and pain in the right knee and arm. Walking was difficult. In spite of these symptoms, the patient went to his office, where he remained until afternoon. On the way home he felt very weak. When he got into bed, he had a chill, lasting twenty minutes, followed by sweating. The headache

continued, and pains developed in the back. Three days after the first chill he had another (October 3) lasting ten minutes. His head and hands became very hot, but he did not sweat. October 4 he felt so weak that he decided to remain in bed. His bowels had not moved since October 1. For several days he had had a dry, hacking cough. In the afternoon, when he entered the hospital, he was chilly and suffered from nausea and vomiting.

Physical Examination. The patient is a well-nourished, well-developed man. Face flushed, skin dry, but in good condition. Tongue large, flabby, coated, and tremulous. Heart and lungs normal. Arteries not thickened. Slight abdominal distention. Slight tenderness in left upper quadrant of abdomen. Liver normal. Spleen not palpable. A few rose spots on the abdomen. The clinical picture is that of mild typhoid fever.

Subsequent History. October 5, headache and abdominal pain all night. Slept about two hours. Fresh crop of rose spots. The spots continued to appear at intervals until the 16th. Slight bleeding when the patient blew his nose. From the 6th to the 9th the patient complained chiefly of vague abdominal pain and headache.

October 9. Conjunctivæ and skin slightly icteric.

October 10. Pain in right hypochondrium (heretofore the pain was not localized). Marked jaundice. Tenderness over gall-bladder. Spleen percusses large, but cannot be felt.

October 11. Jaundice deeper. Tenderness over gall-bladder more marked. Nausea. Severe frontal headache.

October 12. Gall-bladder exquisitely tender. Spasm of overlying muscles. Patient brighter.

October 14. Gall-bladder palpable. Extends one and three-fourth inches below free border of ribs.

October 16. Gall-bladder less tender. Jaundice beginning to clear up.

Thereafter the jaundice gradually disappeared, the gall-bladder receded, and tenderness was developed only on deep pressure. This tenderness persisted, however, until October 27, and for months afterward the patient had a sense of discomfort on bending over. The patient was discharged from the hospital as cured on November 17.

Cytological Examinations of the Blood. October 4. Leukocytes, 5000; polynuclears, 62 per cent.; lymphocytes, 24 per cent.; large mononuclears, 8 per cent.; transitionals, 4 per cent. No malarial organisms.

October 5. Leukocytes, 6000; polynuclears, 77 per cent.; lymphocytes, 14 per cent.; large mononuclears, 8 per cent.; eosinophiles, 1 per cent.

October 9. Leukocytes, 6400; polynuclears, 45 per cent.; lymphocytes, 29 per cent.; large mononuclears, 6 per cent.; transi-

tionals, 18 per cent.; eosinophiles, 5 per cent.; mast cells, 0.3 per cent.

October 10. Leukocytes, 8000; polynuclears, 66 per cent.; lymphocytes, 20 per cent.; large mononuclears, 10 per cent.; transitionals, 2 per cent.; eosinophiles, 4 per cent.

Two blood cultures in broth were made October 5 and 9, but both proved negative (Buxton).

Three rose spots were examined bacteriologically with negative results (Buxton).

The urine was examined bacteriologically twice and found negative (Buxton, Warren).

The feces were examined three times for Bacillus typhosus or any of the intermediates, and also found negative (Norris, Buxton).

AGGLUTINATION REACTIONS.[1] AGAINST BACILLUS TYPHOSUS.

October 9, 10, 13, 14, 16, 18, 19:
Microscopically, in dilution of 1 to 50, negative after two hours.
Macroscopically, in dilution of 1 to 100, negative after twenty-four hours.

October 21:
Microscopically, in dilution of 1 to 50, negative after two hours.
Macroscopically, in dilution of 1 to 100, negative after twenty hours.
Macroscopically, in dilution of 1 to 200, negative after twenty hours.

October 23:
Microscopically, in dilution of 1 to 50, negative after two hours.
Macroscopically, in dilution of 1 to 100, negative after twenty hours.
Macroscopically, in dilution of 1 to 200, negative after twenty hours.
Macroscopically, in dilution of 1 to 400, negative after twenty hours.

October 24:
Microscopically, in dilution of 1 to 50, negative after two hours.
Macroscopically, in dilution of 1 to 200, negative after twenty hours.
Macroscopically, in dilution of 1 to 20, negative after twenty hours.

AGAINST BACILLUS PARATYPHOSUS (TYPES A AND B).

October 14:
Macroscopically, in dilution of 1 to 10, positive after twenty hours.

October 18 and 19:
Macroscopically, in dilution of 1 to 100, negative after twenty hours.

October 21:
Macroscopically, in dilution of 1 to 100, negative after twenty hours.
Macroscopically, in dilution of 1 to 200, negative after twenty hours.

October 23:
Macroscopically, in dilution of 1 to 100, negative after twenty hours.

October 24:
Macroscopically, in dilution of 1 to 200, negative after twenty hours.
Macroscopically, in dilution of 1 to 20, negative after twenty hours.

[1] The dilutions stated are dilutions of the serum before admixture with the suspensions of bacilli—the final dilutions, therefore, would be twice as high.

AGAINST BACILLUS COLI (STRAIN A).

October 14:
Macroscopically, in dilution of 1 to 20, positive after twenty hours.

October 18 and 19:
Macroscopically, in dilution of 1 to 100, positive after twenty hours.

October 21:
Macroscopically, in dilution of 1 to 200, positive after twenty hours.

October 23:
Macroscopically, in dilution of 1 to 400, positive after twenty hours.

October 24, 26, and 28:
Macroscopically, in dilution of 1 to 200, positive after twenty hours.

October 30 (strains A and B):
Macroscopically, in dilution of 1 to 200, positive to A after twenty hours
negative to B after twenty hours.

November 1:
Macroscopically, in dilution of 1 to 100, positive to A after twenty hours.
negative to B after twenty hours.

November 15, the serum in dilution of 1 to 100 was tested against seventeen strains of Bacillus coli, including strains A and B previously used.

The reactions were positive with strains 2, 6, 13, and A.

December 29, the serum no longer agglutinated any strain of Bacillus coli in dilutions of 1 to 100 and 1 to 20.

All of the reactions above noted were end-reactions—tests with more dilute serum proving negative.

While we are aware that we have not proved *beyond doubt* (through our failure to recover the colon bacillus from the blood) whether G. K.'s. infection, was local (that is, a colon bacillus cholecystitis) or general, we are convinced that we have proved that the disease was not caused by Bacillus typhosus or any of the intermediates. The case was considered clinically to be mild typhoid fever by all who saw him, including the late Dr. Loomis and Dr. Thompson. Nevertheless, to forestall criticism, it will be well to discuss the clinical differential diagnosis.

The various clinical possibilities seem to have been: (1) Clinical typhoid fever, complicated by cholecystitis. (2) Primary cholecystitis. (3) Tuberculosis. (4) Sepsis. (5) Pyelitis.

The condition of the urine throughout the disease excludes pyelitis.

The course of the disease and the subsequent history exclude sepsis and tuberculosis (the patient was not lost sight of until the fall of 1907).

There remain only the two varieties of cholecystitis, catarrhal and suppurative. Suppurative cholecystitis may be excluded for the same reasons as sepsis. As against a primary catarrhal cholecystitis caused by the colon bacillus, we base the clinical diagnosis of a typhoid-like fever upon the following facts: mode of onset with the gradually developing malaise, initial chills, slight bronchitis, slight epistaxis(?), persistent headache, appearance of rose spots on the fifth day of the disease, the course of the disease, the emaciation, and the phenomena of convalescence. Leukopenia, with a low polynuclear count, was present until the day after the cholecystitis

became evident clinically. The leukocytes then rose to 8000 and the polynuclears from 45 per cent. to 66 per cent. Though there had been previously vague abdominal pains, these are not uncommon in typhoid fever. Definite tenderness over the gall-bladder did not develop until the day following the subicteric tinging of the conjunctivæ and skin. We are very positive in this statement, because for years it has been our habit to examine daily the gall-bladder region of all patients in the wards suffering from typhoid fever. From all of these considerations we feel justified in thinking that the case of G. K. was clinically typhoid fever, complicated by cholecystitis.

In order to make this study as complete as possible, we have sought during the last three years to obtain the serum of cases suffering from a colon bacillus cholecystitis for the study of its agglutination reactions, but unsuccessfully. We have also written to prominent surgeons to obtain such data, but none of them could furnish the desired information. The literature is silent upon this point. The development of immune bodies for various strains of Bacillus coli in cases of cholecystitis caused by this bacillus offers an interesting and important field for study.

Though it is well established that Bacillus coli may enter the blood during life, only a limited number of cases have been reported in which this fact has been demonstrated by blood culture. Most recent writers on localized colon bacillus infections, for example, of the gall-bladder and of the kidney and its pelvis, recognize the systemic blood stream as one of the channels of infection (Lenhartz, Lockwood, Brewer). While in most of these cases it is probable that the bacillus is only transiently in the blood, Lenhartz and Schmeidemandel have reported cases in which the bacillus has been obtained by blood culture.

A limited number of cases of colon bacillus septicemia have been reported (Bertelsmann and Mann, Lenhartz, Sittman and Barlow, Albarran, Libman, De Gennes and Hartmann, Hitschmann and Michel, Stern, Stadelmann and Wolff-Eisner, Fehling, Blackader and Gillies).

With the possibility of the entrance of the colon bacillus into the blood during life proved, it seems to us that it need excite no surprise that some strains of this bacillus in view of their close resemblance to some of the paracolons, may cause under certain conditions a disease clinically identical with typhoid fever. Buxton and one of us have suggested that even Bacillus typhosus is incapable of causing typhoid fever unless it develops in the general lymphatic system, including the spleen and bone-marrow, and thence invades the blood, where it is destroyed in large numbers. Localized infections with Bacillus typhosus do not cause typhoid fever. Lentz thinks that the same conditions of infection obtain in the typhoid-like types of paratyphoid infection. While there is no direct evidence to offer,

from analogy it is probable that when a colon bacillus causes the symptoms of typhoid fever, the distribution of the bacillus in the body is the same as in the case of typhoid and paratyphoid infections.

Discussion of the Agglutination Reactions for Bacillus Coli. The blood examinations in Cases I and II of the Bellevue Hospital series have been noted in the clinical report. The presence of Bacillus coli in blood taken from a vein leaves no doubt as to the bacteremia, and we feel inclined to accept this as evidence that this bacillus was the cause of the infection. No tests for immune bodies (agglutinins) were carried out.

In Case III, G. K., blood taken from a vein on two occasions proved sterile, and the supposition that Bacillus coli communis was the cause of the infection is based upon the facts that immune bodies (agglutinins) for a certain strain of Bacillus coli were found in the blood during the febrile period and were not found after recovery, and that the same strain of Bacillus coli was not agglutinated by many other sera in high dilution, thus fulfilling our own ideas of the diagnostic value of agglutination reactions and coinciding with the results of Klieneberger.

	Oct. 10, 1905, 1–50 to 1–100.	Oct. 12, 1–100.	Oct. 14, 1–100.	Oct. 21, 1–200.	Oct. 23, 1–400.	Oct. 24, 1–400.	Oct. 26, 1–100.	Oct. 28, 1–200.	Oct. 30, 1–200.	Nov. 1, 1–100.	Nov. 15, 1–100.	Dec. 29, 1–100 to 1–20.
Bacillus typhosus.	—	—	—	—	—	—	..	..	..	..	—	—
Bacillus paratyphosus A and B.	..	..	—	—	..	..	..	..	..	..	—	—
Bac. coli A.	..	..	++	+++	+++	+++	+++	+++	+++	+++	+	—
Bac. coli B.	..	..	—	—	..	..	..	..	—	—	—	—
Bac. coli No. 2, No. 6, No. 13.	..	..	..	..	..	..	..	..	..	..	+	+
Bacillus coli, 12 strains.	..	..	..	..	..	..	..	..	..	..	—	—

Dilutions refer to the diluted serum; final mixtures were twice the serum diluted.

Agglutination tests in Case III gave the following results: October 10 and 12, tests against Bacillus typhosus were negative in dilutions 1 to 50 and 1 to 100. October 14, the serum diluted 1 to 100 was tested against Bacillus typhosus, Bacillus paratyphosus (A and B), and one strain (A) of Bacillus coli, and the reaction for strain A Bacillus coli was positive; for the other organisms, negative.

Control tests were carried out with strains of Bacillus typhosus, Bacillus paratyphosus, and certain negatively reacting Bacillus coli strains, and the reactions noted were end-reactions—tests with more highly diluted serum proving negative.

The macroscopic method, with fresh emulsions prepared after Borden's method, was used, and also fresh suspensions in 0.8 per cent. sodium chloride solution. October 21, the serum diluted 1 to 200 gave the same positive reaction against strain A Bacillus coli. October 23, the serum diluted 1 to 400 was positive for A. October 24, 26, and 28, serum diluted 1 to 200 was positive.

October 30 the serum was tested against strains A and B Bacillus coli and proved positive to strain A and negative to strain B, in dilution 1 to 200. November 1, the same reactions as on October 30 were obtained in dilutions of 1 to 100.

November 15, the serum diluted 1 to 100 was tested against 17 strains of Bacillus coli, including strains of A and B used previously. The reactions were positive with strains 2, 6, 13, and A. December 29, the serum no longer agglutinated any of the Bacillus coli strains in dilutions of 1 to 100 and 1 to 20.

Bacillus coli strain A which constantly agglutinated with the serum diluted 1 to 100 from October 14 to November 15, and 1 to 400 October 23, had been obtained from one of the Bellevue cases above cited (1903). Strains 2, 6, and 13, which the serum, diluted 1 to 100, agglutinated November 15, had been obtained from the blood and the stool of a case of colon bacillus septicemia. Strain 6 was obtained from the ureter of a case of pyelitis. Therefore the serum of G. K. agglutinated 4 strains of Bacillus coli obtained from three different individuals.

For the various strains of Bacillus coli and Bacillus paratyphosus we were indebted to the laboratories of Drs. Buxton and Elser.

In connection with the study of Case III we have made numerous tests of the serum reactions of non-icteric and icteric cases.

Non-icteric Cases. During the typhoid seasons of 1905, 1906, and 1907, 100 serum tests on 54 cases were carried out against Bacillus coli (strains A, B, and 2) and Bacillus paratyphosus (strains A and B).

The serum of a normal individual, used as a control, and the sera from 52 febrile cases, many of them Bacillus typhosus infections, were found negative to Bacillus coli (strain A) in dilutions above 1 to 20, with one exception. The serum from one febrile case, diluted 1 to 20, agglutinated Bacillus coli (strain A), and the serum from two cases, diluted 1 to 20, agglutinated Bacillus paratyphosus strains, but did not agglutinate Bacillus typhosus or Bacillus coli strains.

The sera from 31 cases did not agglutinate any one of several strains of the typhoid-colon group.

The sera from 16 cases agglutinated Bacillus typhosus, but not Bacillus paratyphosus and Bacillus coli strains.

Unfortunately, in 40 cases single tests only were made: In 6 cases two tests; in 3 cases three tests; in 1 case four tests; in 1 case six tests; in 2 cases nine tests; in 1 case ten tests; in the cases with six, nine,

and ten tests the blood was taken at intervals extending over an entire month.

CASES WITH JAUNDICE AS A SYMPTOM. The sera of 13 cases with well-marked jaundice as a symptom were tested against strains of Bacillus typhosus and Bacillus coli—single observations in 12 cases and three observations in 1 case. In one instance, a single observation, Bacillus typhosus was promptly agglutinated both microscopically and macroscopically; in a second instance, Bacillus coli (strain B from a pleural exudate) was agglutinated macroscopically in dilution of 1 to 100, but not above; in a third instance, a case of catarrhal jaundice, Bacillus coli (strain 2) was agglutinated with serum diluted as high as 1 to 100.

Thus, none of these cases, with or without jaundice, agglutinated any one of the typhoid-colon group which were tested in dilutions above 1 to 100. In view of the fact that the sera from three jaundice cases were not tested over a period of weeks, we do not intend to discuss these single positive reactions as evidence that a particular strain of Bacillus coli was the infecting agent, although Kelly's paper suggests that such reactions may be of value in this direction.

For Bacillus typhosus it is well known experimentally that the sera of immunized animals must be diluted to the thousands before selective reactions are obtained, while in clinical medicine dilutions of the patients' sera 1 to 50 give diagnostic reactions within certain time limits.

In discussing the agglutination tests for the purpose of diagnosticating a colon bacillus infection, the following types of cases must be considered from theoretical and practical standpoints:

A. Cases of undoubted colon-group infection, in which blood cultures were positive during life and not during the pre-lethal stage or post-mortem. These cases may be divided into two groups:

1 (*a*) Cases which were typhoid-like in course, whose blood contained agglutinins for strains of the colon bacillus.

(*b*) Cases which were typhoid-like, whose blood did not contain agglutinins for strains of Bacillus coli.

2 (*a*) Cases which were not typhoid-like in course, whose blood contained agglutinins for strains of Bacillus coli.

(*b*) Cases which were not typhoid-like in course, whose blood did not contain agglutinins for strains of Bacillus coli. Endocarditis and pyelitis are examples of the last two groups.

B. Cases of supposed colon-group infection, with negative blood cultures. These cases likewise may be divided into two groups:

1. Cases which were typhoid-like in course, whose blood contained agglutinins for strains of Bacillus coli.

2. Cases which were not typhoid-like in course, whose blood contained agglutinins for strains of Bacillus coli. Cholecystitis and pyelitis are examples of this last group.

In the light of our knowledge of group-agglutinins, one must con-

sider the relation of other typhoid-colon group infections to infections by the colon bacillus; for example:

1. Cases of undoubted Bacillus typhosus infection, clinically typhoid-like in nature, in which blood cultures and agglutination tests for Bacillus typhosus were positive and agglutinins for colon strains were found.

2. Cases in which agglutinins were positive for Bacillus typhosus alone, with blood cultures positive for Bacillus coli; or in which blood cultures were negative, and yet agglutinins were found for both typhoid and coli strains.

3. Cases in which blood cultures were positive for Bacillus paratyphosus and agglutinins were found for paratyphoid and colon strains.

4. Cases in which blood cultures were positive for Bacillus coli and whose serum agglutinated typhoid or paratyphoid strains.

Finally, the agglutination of colon strains by normal sera must be considered. The results of Klieneberger show a high agglutinin content for normal serum, diluted 1 to 2000 to 3000, for certain colon strains, and would lead one to consider the possibility that some individuals are producers of antibodies for Bacillus coli. Our results are at variance with Klieneberger's in that normal serum and sera from febrile cases have not caused agglutination of colon strains excepting in dilution of 1 to 20 in a febrile case without jaundice, and in dilution of 1 to 100 in two cases with jaundice. This contradiction is readily explained by the fact that in testing 17 different strains of Bacillus coli we found 4 strains only which were agglutinated by 5 out of 67 sera.

The fact that Klieneberger worked with a strain which was agglutinated by many normal sera would not invalidate our results unless it were proved that his colon strain is identical with one or more of those used by us.

Terminal colon bacillus infections and the reactions of typhoid bacillus carriers do not concern us.

Articles in the literature dealing with agglutination tests in Bacillus coli infections are not numerous, yet one or two of them, together with the results obtained in Case III cover the theoretical and practical considerations in respect to diagnostic reactions.

Klieneberger refers to Pfaundler's statement that the colon bacillus is agglutinated when one mixes together the serum and a strain of Bacillus coli obtained from the same patient, and not when the serum and bacteria are obtained from different individuals under normal conditions. From the work of Wassermann and others it is known that immune coli sera differ not only in respect to the strain used for immunizing, but also according to the type of animal used for immunization, and to explain these differences extensive variation in receptors of different coli strains are supposed to exist. Noting these statements, Klieneberger worked with sera from

normal adults, and then from the newborn, with immune coli sera from immunized animals and from a man infected with Bacillus coli. The standard strain of Bacillus coli used was from the urine of a patient with a genito-urinary colon bacillus infection. Thirteen other strains of Bacillus coli were selected from 21 strains, for carrying out absorption experiments on sera agglutinating the standard strain. From six newborn infants, the sera being tested against 6 coli strains, 2 sera agglutinated 2 different strains, one each as high as 1 to 160; and the agglutination from 1 strain only was proved by absorption tests. From normal adults with no history of icterus or typhoid fever, 2 sera, diluted 1 to 1280, agglutinated 2 strains of Bacillus coli; 4 cases, 1 to 320, agglutinated 3 strains; 1 case, 1 to 640, agglutinated 1 strain. It is noticeable that 2 strains were agglutinated by 5 sera diluted 1 to 320 or over, and that each serum, 1 to 320, agglutinated at least 1 strain. Three strains were not agglutinated by any serum, and 5 strains were not agglutinated by any serum diluted above 1 to 80. No serum agglutinated Bacillus typhosus or Bacillus paratyphosus (A and B) above 1 to 20. One normal serum (M) agglutinated weakly 2 strains, with serum dilutions of 1 to 2560; 1 strain, with serum dilutions 1 to 640; 1 strain, 1 to 320; 1 strain, 1 to 160; and 3 strains not above 1 to 40. With normal serum M, by precipitation with 8 strains, partial agglutinins were found for 5 colon strains, and with normal serum R, partial agglutinins for 4 colon strains were found; and, further, it was found that one could group the agglutinable strains under two subdivisions, one of which was agglutinated constantly by highly diluted sera and the other irregularly and by low dilution. A second group of coli strains was not agglutinated by any serum. From these observations Klieneberger concludes that a tested strain of Bacillus coli has set up a specific immunity only when we can show that it is agglutinated with abnormally high dilutions, or that normal serum does not agglutinate the strain. He also speaks of an increment in agglutinating power as indicative of immunization from active infection, and cites 2 cases as showing this fact; and he refers to observations on Bacillus coli infections of the urinary tract, during which he had noted the same incremental auto-immunization.

In opposition to Pfaundler, the first fact noted was that in many Bacillus coli infections an agglutinating effect for the causative strain was absolutely wanting. He obtained a "group" agglutination for the typhoid bacillus four times, and for the paratyphoid bacillus once, with the Bacillus coli serum. Klieneberger concludes that in cases of Bacillus coli infection in man, when the causative agent has not been isolated, it is scarcely possible to prove a Bacillus coli infection with serum tests unless immunity to Bacillus coli can be determined, when an increasing agglutination and abnormally high agglutination is found, and normal controls are negative. Further, Klieneberger immunized rabbits against 5 active strains

of Bacillus coli with 1 c.c. of a formalized broth culture injected intravenously and obtained a high agglutinating power against the immunizing strain and a much less ($\frac{1}{20}$ of the high) agglutinating power against 3 non-immunizing strains, and negative results against 8 other Bacillus coli strains, 1 typhoid strain, and paratyphoid strains A and B. A similar chief agglutinin was obtained with the 5 strains used for active immunization.

Case III (G. K.) illustrates well the increment of agglutination which is essential, according to Klieneberger, for the proof of Bacillus coli immunization.

Mayer found one strain (R) which was usually agglutinated by normal serum diluted 1 to 100 (macroscopic test) and 1 to 250 (microscopic test), and other strains (M, N, L) with which agglutination was not obtained with sera little diluted. Strain M not only did not agglutinate well with normal serum, but was not readily agglutinated by serum (1 to 50) from an animal immunized to strain R, and did not produce agglutinins of any power when used for immunization.

This variation in the agglutinability and in the antigen for agglutinin in certain strains was the most interesting result of Mayer's investigation. Dead bacteria (Bacillus coli, twenty-hour cultures) gave stronger agglutinins and with more certainty than living bacteria when used for immunizing animals, an observation also made by Radzievsky (1900).

Bauermeister's case of general Bacillus coli infection, and later abscess of the testicle, was not carefully studied for agglutinins. An organism isolated from the abscess gave a "Widal" (?) 1 to 50, while the bacilli (Bacillus coli) from the blood and from the urine were not agglutinated; whether with the patient's serum or not does not seem clear. The bacillus isolated from the abscess—one and one-half years after the observations on the blood and urine—did not present the same cultural characteristics as those of the bacilli isolated from the urine and blood.

Jochmann, in 1906, recorded some interesting observations on infections of the urinary tract and general infection with the colon bacillus—two cases with an intermittent fever. In one case, following operation for carcinoma of bladder and subsequent catheterization, pneumonic signs developed, and later chills occurred, after which blood cultures with 20 c.c. of blood gave a growth of 10 colonies of Bacillus coli. In a second case, during a cystitis due to Bacillus coli, 8 colonies of Bacillus coli were grown from 20 c.c. of blood. Jochmann considered both these cases a blood invasion by Bacillus coli. In the first case Jochmann tested the agglutinating power of the serum against Bacillus typhosus, Bacillus paratyphosus, and several Bacillus coli strains. No reactions were obtained with Bacillus typhosus and Bacillus paratyphosus, and one strain of Bacillus coli was agglutinated by serum diluted 1 to 80, and the

Bacillus coli isolated from the patient was agglutinated by serum diluted 1 to 160. The patient's strain of Bacillus coli was agglutinated also by sera diluted 1 to 80 from four cases of acute cholelithiasis and by serum, diluted 1 to 320, from a protracted case of cholelithiasis; and a case of icterus with hepatic conditions and cholangitis gave a serum which, diluted 1 to 160, agglutinated the same strain of Bacillus coli. These cases he considered Bacillus coli infections. Jochmann tried 8 normal sera against the Bacillus coli strain from his Case I, and obtained reactions with dilutions no higher than 1 to 40. He thought a reaction with serum diluted 1 to 80 necessary to indicate infection with Bacillus coli, and that the reaction would be of value when no group agglutinins for Bacilli typhosus and paratyphosus were obtained. If the latter were present, one might still obtain a specific Bacillus coli reaction by saturating the sera (by absorption) after Castellani's method. Later, Jochmann tested the sera of 16 other Bacillus coli infections and obtained reactions with highly (?) diluted serum, so that he disagreed with Köhler and Scheffler, who claimed that such reactions are of no value. Jochmann also concluded that the homologous strain would agglutinate with more highly diluted sera than the heterologous strains, as stated by Pfaundler, which is contrary to the experience of Klieneberger and to our results.

Fehling's article deals mainly with infections of the genito-urinary tract during pregnancy and the puerperium. In one case of pyelonephritis in pregnancy, with fever for the fourteen days *ante partum* and at delivery, cultures from the blood were sterile; and cultures from the fœtus were sterile. The fever disappeared on the fourth day of the puerperium, and the bacteruria persisted. Fourteen days after delivery the mother's blood, diluted 1 to 150, agglutinated a bacillus (Bacillus coli) isolated from the urine. He concluded that the Bacillus coli was in the mother's blood at the same time, admitting the possibility of agglutinating substance coming from the fœtus through the placenta. From another case, following an obstetrical operation, he obtained Bacillus coli from the blood, urine, sputum, and sweat, and this case he considered the eighth of pure Bacillus coli sepsis recorded in the literature. No agglutination tests were carried out.

The nosological position to be given to the typhoid-like cases caused by Bacillus coli communis demands brief consideration. As in the case of typhoid-like infections by the intermediate members of the Typhaceæ, it is impossible to differentiate these cases from infections by Bacillus typhosus by clinical methods alone. The assistance of the laboratory is required. Before the development of exact bacteriological diagnosis, all typhoid-like cases, by whatever member of the group they were caused, were considered as typhoid fever. The diagnosis was based necessarily upon clinical data. Since the discovery that the symptom-complex of typhoid fever

may be caused by several members of the Typhaceæ, the nosological position of these cases has been under discussion.

Two courses are open to us: to classify the cases clinically or bacteriologically. The former course is the simpler, and is followed in the case of many other diseases. Classification on a bacteriological basis necessitates the introduction of a separate term for each variety of infection. Therefore, we are inclined to advocate the classification of all cases of infection by the Typhaceæ, *when they pursue a typhoid-like course,* as clinical typhoid fever.

CONCLUSIONS. 1. Isolated reactions against Bacillus coli are without significance in respect to the cause of infection.

2. The blood serum of a case of infection by Bacillus coli communis may contain agglutinins for certain strains of this group while the infection persists and not upon the patient's return to health.

3. The detection of an increment of agglutinins in the blood serum for a particular strain of Bacillus coli communis is evidence of a local or general infection with that strain.

4. Some strains of Bacillus coli communis are capable of causing a disease clinically identical with typhoid fever.

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For other references to colon bacillus sepsis, see Jochmann's paper.

ACUTE SYPHILITIC DISEASE OF THE SPINAL CORD.[1]

A DISCUSSION OF "SYPHILITIC SPINAL PARALYSIS."

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YEAR by year it becomes more firmly established that syphilis is the chief cause of acquired disease of the nervous system. The discovery of the spirocheta and the determination of the specificity of the serum reaction for syphilis have given firm foundation to what was previously presumption and conjecture regarding the dependency of certain diseases of the nervous system upon syphilis. Diseases that had been proved statistically to follow almost invariably in the wake of syphilis, like tabes and general paresis, but which some have contended were not syphilitic diseases because the lesions were not "characteristic" of syphilis, give a specific serum diagnostic reaction, and specific antibodies are demonstrable in the serum or cerebrospinal fluid, by a technique which as yet is unfortunately complicated.

There has been much discussion as to the manner in which syphilis attacks the spinal cord and as to whether or not there are different well-defined syphilitic diseases of that organ aside from tabes, which is now universally spoken of as a parasyphilitic disease. In 1892 Erb attempted clinically to differentiate a form of disease of the spinal cord which occurred exclusively in individuals who had previously suffered from syphilis and which resembled spastic spinal paralysis, differing from it, however, in a definite and charateristic way. It was characterized clinically by the triad of symptoms of spastic spinal pralysis *plus* some disturbance of the function of the bladder and slight subjective and objective sensory disturbance. He maintained that the occurrence of symptoms other than those mentioned made the diagnosis doubtful. The onset of the disease is usually slow and insidious, but sometimes more rapid, and improvement and long periods of standstill are possible. When Erb published his description of the disease no postmortem evidence from typical cases was obtainable. It seemed to him that the anatomical basis of the disease should consist of partial, symmetrically situated lesions in both halves of the cord in the dorsal region,

[1] Read at a meeting of the New York Academy of Medicine, October 1, 1908.

chiefly in the posterior lateral tracts, with partial involvement of the gray posterior columns and the white posterior tracts. He was unwilling to say positively what the nature of the lesion was, whether specific syphilitic infiltration, syphilitic infection of the vessels, or degenerative change. There was much corroboration of the existence of the disease from physicians in this country and aboard, but there was likewise much opposition to the acceptation of it, many maintaining that the disease was one that had long been recognized and was merely a gummatous myelitis, syphilitic disease of the vessels, or syphilitic meningomyelitis.

There have been nine or ten cases of typical syphilitic spinal paralysis that have come to autopsy since the publication of Erb's original paper. These cases are those of Westphal,[2] Eberle,[3] Nonne (2 cases),[4] Williamson,[5] Dreschfeld,[6] Long and Wiki,[7] Wimmer,[8] and Renner.[9] The first cases that were investigated seemed to show that the lesion was that of a combined system disease, that is to say, a primary gray degeneration, in various fiber systems of the spinal cord, chiefly in the posterior half of the lateral tracts, the direct cerebellar tracts, and the tracts of Gowers; somewhat also in the posterior tracts, those of Goll and Burdach.

Nonne, ten years later, in 1902, demonstrated most convincingly that although syphilitic spinal paralysis was clinically a well-defined entity, it had by no means a constant, unvarying, morbid anatomy. He showed that the anatomical foundation of the disease might be: (1) A chronic, punctate, transverse myelitis, with ascending and descending degeneration; (2) a similar condition associated with primary degeneration of the pyramidal tracts; (3) degeneration of the pyramidal tracts alone; or (4) degeneration of the posterior and of the lateral columns (pyramidal tracts, direct cerebellar tracts, Gowers' tracts), a combined systemic disease. His study further showed that although the bloodvessels and the meninges may be diseased in syphilitic spinal paralysis, they may also be free from disease.

In a recent contribution of Nonne to the subject his contention has been further substantiated, as it has likewise been by the case of Wimmer and our own case.

The last case which Nonne published was that of a man who had had syphilis at thirty followed by vigorous mercurial treatment. When fifty-four years old he began to complain of difficulty in walking, slight radiating pains in the legs, and hesitancy in urination. Examination showed spastic gait, increased tendon jerks, slight

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[7] Nouvelle Iconographie de la Salpêtrière, 1902.
[8] Deutsche Zeitschrift f. Nervenheilkunde, 1907, vol. xxxii.
[9] Ibid., vol. xxxiv, p. 451.

ankle and patellar clonus, normal skin reflexes, some diminution of pain sensibility in irregularly distributed areas, and loss of the light reflex of the pupils. The cord showed a very slight punctate, chronic myelitic degeneration of the dorsal segments without secondary ascending or descending degeneration, but with very slight but unquestionable degeneration in Goll's columns of the cervical cord and the upper dorsal cord, as well as degeneration of the pyramidal tracts in the lumbar cord. In addition to these there was thickening of the bloodvessels without specific character, chronic endarteritis of the anterior spinal artery, and indefinite thickening of the meninges over the posterior part of the cervical and dorsal cord.

In 1902 Long and Wiki published their study of a case in which there was, in addition to punctate myelitis in the dorsal region, a degeneration of the lateral pyramidal tracts, direct cerebellar tracts, and Goll's column, and acute arteritis. The authors considered the tract degeneration to be secondary to the punctate myelitis, but Erb, in commenting upon the case, maintained that the main factor was a combined system disease with chronic sclerosis of the bloodvessels, by the side of which the rather diffuse chronic myelitic lesions in the dorsal cord appear etiologically as a quite permissible complication.

One of the most important cases that has so far been published is that of Wimmer. A man who had complained of periodic jabbing pains in his legs from the time he was thirty years old, became infected with syphilis when forty-five years old. He took active mercurial treatment, but eighteen months after the infection he developed typical syphilitic spinal paralysis, which ran a rapid course. About six months after the onset of the disease he developed cystitis and pyelonephritis, and died in a state of septicemia. The spinal cord showed the following lesions: (1) A focus of disease in the eighth and to a slight degree the ninth dorsal segments. (2) Slight meningeal changes in the dorsal and cervical cord. (3) Extensive bloodvessel affection. (4) Fairly extensive degeneration of Goll's column, the direct cerebellar tracts, a part of Gowers' bundle, and also the anterior uncrossed pyramidal tracts.

The case related by Renner is by no means typical of syphilitic spinal paralysis, and it should not be included in the category; the occurrence of optic atrophy and of ataxia of the upper extremities makes it impossible to include it in this category, but rather in that of tabes.

CLINICAL SUMMARY. *A youth aged twenty-three years, addicted to the intemperate use of spirits, became infected with syphilis September,* 1907. *Cutaneous and mucous membrane manifestations developed promptly. December,* 1907, *he had aphonia, due to syphilitic laryngitis. Six months after infection he developed weakness, stiffness and paresthesia of the lower extremities, and slight difficulty in urinating. The weakness of the legs intensified rapidly up to moder-*

ate paraplegia. This was promptly followed by retention of urine and later by incontinence of urine and feces, and impotency. After a very brief time he recovered sufficiently to leave the hospital, the symptoms at this time being those characteristic of syphilitic spinal paralysis. Cystitis and surgical kidney developed rapidly and he died eight months after infection, during which time he had been given mercury and iodide of potassium.

The patient, a young man aged twenty-three years, by occupation an actor, entered the City Hospital March 29, 1908, complaining of inability to walk, of numbness in the legs, of difficulty in discharging the urine and controlling the feces, and of huskiness of the voice. These symptoms, save the huskiness of the voice, had existed about a fortnight.

The mother, a paternal uncle and aunt had died of tuberculosis at an early age. The patient had been for several years addicted to the use of beer, whiskey, and absinthe. In August, 1907, he had an attack of gonorrhœa which lasted three months. In September, 1907, he became infected with syphilis, and in October a rash appeared upon the skin and mucous membranes. In December his voice became husky and soon he became unable to speak above a whisper.

Early in March, 1908, on the appearance of the nervous symptoms enumerated above, the aphonia suddenly disappeared. March 3 he received mercury hypodermically; the next day he complained of a numb sensation in the right leg, which he attributed to the injection. On March 6 he noticed weakness of the right leg. Next day it was difficult for him to walk or to stand, both legs being weak. At the end of the third day he was so paraplegic, he says, that he could not move the feet, legs, or thighs. Coincident with the occurrence of this motor paralysis he complained of a sense of numbness throughout the lower extremities, more noticeable in the hip than in the legs or the thighs. From March 6 to 9 he was unable to pass water, and it had to be withdrawn by catheter; after that he became incontinent both of urine and feces, but two weeks later retention again developed, and it became necessary to use the catheter again. A week after the onset of the paraplegia a bedsore formed over the sacrum.

He was quite positive that previous to the onset of the paralysis there had been no symptoms indicative of brain or spinal cord disease, no headache, dizziness, disturbance of function of the special senses, nor pain.

March 24, 1908. On the patient's arm and back a fading syphilitic eruption is still to be seen. On either side of the neck is a chain of enlarged indurated lymphatic glands. Over the sacrum is a bedsore about as large as the hand. The patient cannot stand, and he cannot raise himself in bed save by the assistance of the arms. The lower extremities can be flexed and extended, abducted and

adducted, all feebly and with considerable spasticity. The tendon jerks of the lower extremities are lively. The Babinski and the Oppenheim phenomena are elicitable in both lower extremities. Tactile sensibility is very much blunted in both feet and ankles, and there is slight hyperesthesia of the legs. The temperature sensibility is likewise diminished in the feet and legs, but pain sensibility is preserved. There is no atrophy of the lower extremities. The upper extremities are unaffected. There are no pupillary disturbances and no involvement of any of the cranial nerves.

Bichloride of mercury was administered hypodermatically and attention was given to the bladder and bowels. Examination of the urine made on many occasions showed the presence of a faint trace of albumin, a considerable amount of pus, many epithelial cells, and a diminution of the total solids. Examination of the blood showed an approximately normal condition, with an increasing number of leukocytes. When he left the hospital, on April 28, he had made considerable improvement; the bedsore had very nearly healed, and he was able to walk with the aid of a stick; his gait, however, was typically spastic, but the muscular rigidity was comparatively slight.

While out of the hospital he received an injection of salicylate of mercury and 120 grains of iodide of potassium daily. On April 30 he complained of sensations of weight in the stomach, of nausea, and of vomiting; likewise of a sensation of tenderness of the spine, as though he had been beaten. From that day until his reëntrance to the hospital, May 4, headache and dizziness were also distressing symptoms.

Examination at this time showed very similar conditions to those existing when he left the hospital. He was able to walk without assistance, the gait being one of enfeeblement and spasticity. The power of flexion and extension of the legs and thighs was very little impaired. All the tendon jerks of the lower extremities were increased, the Babinski phenomonon and ankle clonus were present. The abdominal and cremasteric reflexes were not elicitable. The upper extremities and cranial nerves showed no abnormalities. The lower extremities were slightly spastic.

Examination of the sensory state of the lower extremities showed postural sensibility normal, tactile sensibility very slightly obtunded in the feet, and thermal sensibility was more impaired than any other form. On the legs he did not readily distinguish warm from hot objects, and unless the object was very hot he did not distinguish heat from simple touch. In the thighs, however, warm and hot were distinguishable all over, and cold readily. There was slight ataxia of the legs, most manifest when he attempted to put the heel of one foot on the knee of the other leg.

On May 7, 1908, that is three days after his readmission, he developed symptoms of septic pyelitis, septic infarct, pain in the left

loin particularly on micturition, pinched expression of the face, profuse perspiration over the entire body, feeble pulse, and elevation of temperature. Cystoscopic examination showed the mucous membrane of the bladder much injected, its folds considerably swollen. The ureters were not catheterized. Rectal examination was negative save that it showed the sphincter to be of fairly good tone.

On May 20 nephrectomy of the left kidney was performed, which the patient did not long survive.

SUMMARY OF PATHOLOGICAL CHANGES. *Areas of softening, principally in the posterior half of the cord on transverse section, extending from the first to the ninth dorsal segments, associated with changes in the bloodvessels, thickening and round cell infiltration of the vessel walls, circumvascular exudation, vascular thrombosis and hemorrhages; syphilitic myelitis; sclerotic patches due in part to growth of glia cells, principally toward the periphery of the cord, especially of the posterior and posterior lateral tracts; secondary degeneration of the posterior columns, especially the column of Goll and also of the direct cerebellar tract and Gowers' tract in the upper thoracic and cervical regions; slight, but distinct, productive meningitis, patch-like distribution in many thoracic segments. The cells of Clarke's column much degenerated; those of the anterior horn suffering to a much less extent, possibly the effects of sepsis.*

When the spinal cord was removed the thoracic portion was noted to be of less firm consistency than normal, and when cut across, reddish brown areas, resembling hemorrhagic foci, were seen macroscopically. These were more striking after the cord had hardened. On sectioning the cord these areas of softening appeared and disappeared, having a wide distribution, from the first to the ninth dorsal. They were, however, most conspicuous in the mid-dorsal region, and were situated largely in the interior of the cord, in the middle and the posterior thirds. They involved, at different levels throughout the diseased portion, the posterior columns, the posterior horns, and the lateral columns in the region of the posterior horns, also the gray matter in the vicinity of Clarke's column, the lateral horns, and in one instance the anterior horns. There were about seven of these areas in all, most of which did not extend through more than one segment of the cord, the greatest extent being three segments. Many sections through the affected region did not present these softened areas.

Examined after appropriate hardening and staining, the vascular alterations are most striking, veins, arteries, and capillaries all being involved. The branches of the posterior spinal vessels are more uniformly affected, both the large and the small, the meningeal and the medullary; but over a small extent the anterior vessels are as seriously diseased as the posterior.

Many vessels exhibit marked endarteritis, distinctly hyaline in character, with more or less narrowing of their caliber. This is a

prominent feature of the capillaries in the cord substance, where, in certain areas of softening, minute hemorrhages have invaded the surrounding tissues and red blood cells have migrated far from their source. These hemorrhages are to be seen in the gray matter, especially in the region of Clarke's column, in the posterior columns, and to a less degree in the lateral columns (Fig. 1). One of these areas, circular in outline and situated at the ventral end of the posterior column, is riddled with erythrocytes, but no bloodvessels from which they might have come is discernible. In close proximity to these thickened capillaries are other thin-walled capillaries, the larger of which lie in wide perivascular spaces and are enormously engorged to the size of the large meningeal arteries; the smaller,

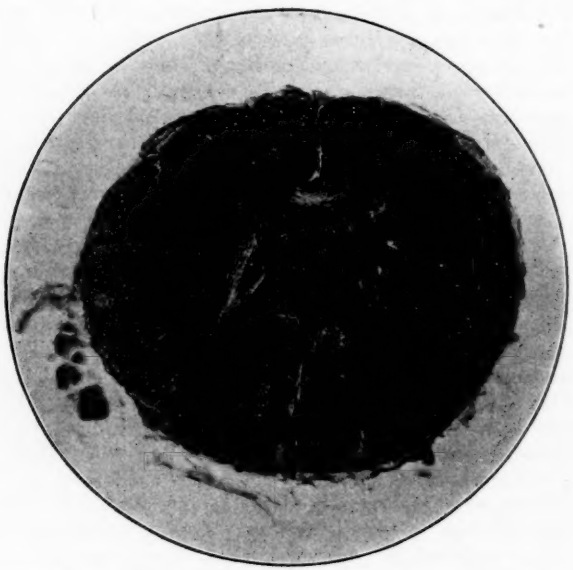

FIG. 1.—Dorsal cord showing hemorrhages in the region of Clarke's column, the posterior columns and the lateral tracts, and the secondary degeneration.

poorly formed and more numerous, are budding bloodvessels. In those sections which show the changes in the anterior gray matter of the cord, the artery of the anterior fissure and the ventral meninges are thickened, and show slight hemorrhages. Perivascular thickening is frequently encountered, and there is extensive proliferation surrounding many meningeal vessels.

The circumvascular cellular infiltration, which is very conspicuous, is significant of the acuteness of the process (Fig. 2). Comparatively few vessels, large or small within the stated limits of the lesion escape

this round-cell infiltration, and many of the vessels in the cervical and lumbar regions show it to a marked degree. It is noticeably more frequent, however, in the posterior half of the cord, where many vascular spaces are widened; and tortuous vessels, running from the periphery, especially those cut longitudinally, show varying numbers of round cells lying both in their walls and in their circumvascular lymph spaces. Meningeal vessels frequently display profound cellular invasion, which, uniformly, has its origin in the proliferated outer coat, and may ultimately involve the entire wall, so that occasionally a vessel, solid with round cells, is wholly obliterated (Fig. 3). Vessels in the vicinity of those seriously diseased show comparatively minor changes, and there is no ratio between the thickening and the infiltrating processes.

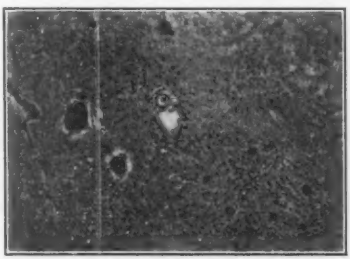

FIG. 2.—Circumvascular cellular exudation and thrombosed vessels.

The round cells are of the lymphocytic variety, having a thin layer of protoplasm surrounding a nucleus, fairly regular in outline which stains black. In the cord these round cells are almost wholly confined to the bloodvessels; in the meninges, however, they infiltrate the tissues, seeming to travel along the lymph spaces (Fig. 4).

These vascular alterations, evidently syphilitic, are of a true inflammatory character. They and the pathological alterations, which condition patchy softening in the parenchyma and the glia tissue of the cord, constitute a true syphilitic myelitis.

The areas of softening are evidently of recent origin, and while some of them show budding bloodvessels, the glia replacement is only of a moderate degree. The cells are of two types, the normal having an opaque nucleus staining more darkly than the other; it is larger, more definitely granular and translucent, varies in outline, and shows a tendency to disintegrate. The destruction of the nervous elements, in most cases only partial, is characterized by swelling of the axones, bulging and disintegration of the myelin sheaths, with ultimate disappearance of both nervous elements, large perforations remaining. It is not uncommon to find intact axis cylinders persisting after complete destruction of the medullary sheaths.

Small patches of degeneration, made conspicuous after staining and showing decided preference for the posterior columns and the

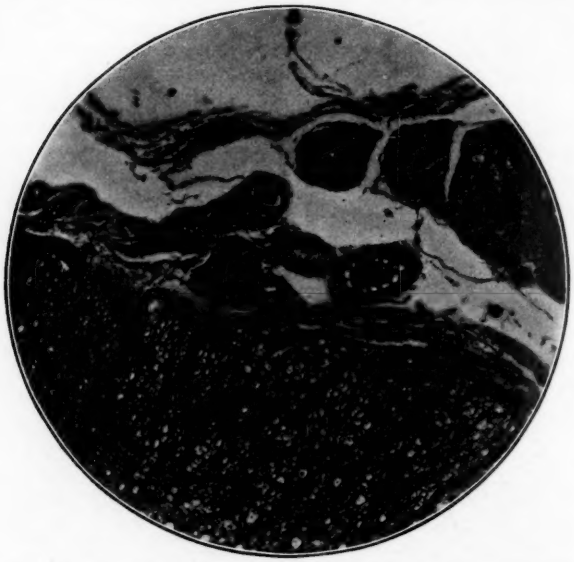

FIG. 3.—Profound involvement of meningeal vessels and thickened meninges.

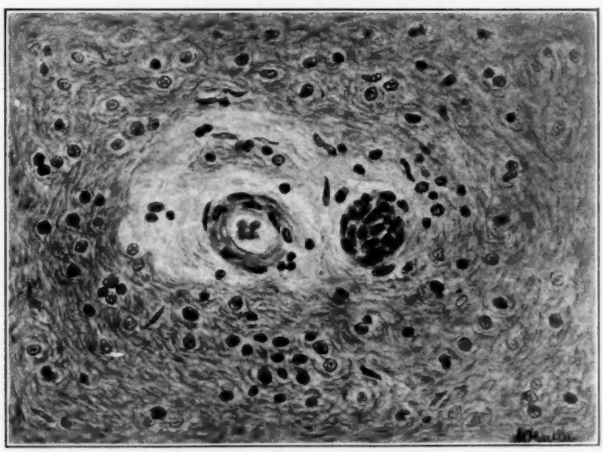

FIG. 4.—Area of softening; illustrating inflammatory character of the vascular alterations.

posterior half of the lateral columns, border the periphery of the cord like dipping pia, radiate more deeply into its substance, and lie

isolated almost anywhere in its interior (Fig. 5). Some of these patches are sharply defined, and these frequently follow the course of the bloodvessels. Others are more or less devious and irregular in outline. In keeping with the size and number of these evidently primary foci, there is destruction and disappearance of the nervous elements.

On cursory examination, the pia seems to be only slightly diseased, but careful examination shows numerous well-defined patches that are moderately thickened from true proliferation and more or less infiltrated with round cells. These areas are found in both the

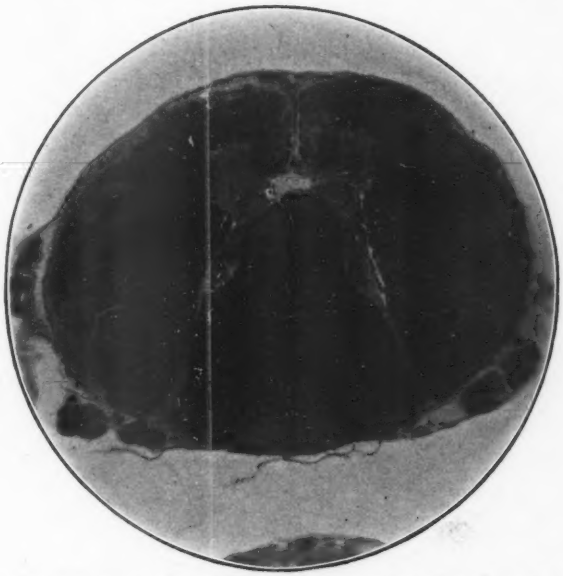

FIG. 5.—Patches of degeneration of irregular distribution, but principally of the posterior columns, and thickening of the meninges.

anterior and posterior surfaces of the cord, mostly in the posterior, and to a much less extent in the lateral aspects. Hemorrhages are also seen in the meninges (Fig. 6). The dura was intact.

As might be expected, the ganglion cells appear to be well preserved except in the vicinity of the lesions noted above. In these regions the cells of Clarke's columns show frank degenerative processes of varying degrees from slight central chromatolysis to complete chromatolysis, eccentric, shrunken, or absent nucleus, and atrophy of the cell. To a less extent, the lateral and anterior horn cells share in these changes (Fig. 7). The secondary degenerations are enumerated in the summary, and are depicted in the illustrations.

COMMENT. We do not maintain that this is an example of syphilitic spinal paralysis. It does not conform to that disease either in its onset or in its early symptomatic display. A slow and insidious onset is considered quite characteristic of the disorder which Erb described. In the case that we report the onset was abrupt and paraplegia developed within a few days; but the point to which we particularly wish to draw attention is that this paraplegia soon disappeared and the patient left the hospital. When he left the hospital he was clinically an example of syphilitic spinal paralysis. He had spastic gait, increased tendon jerks, muscular hypertonus, sensory disturbance

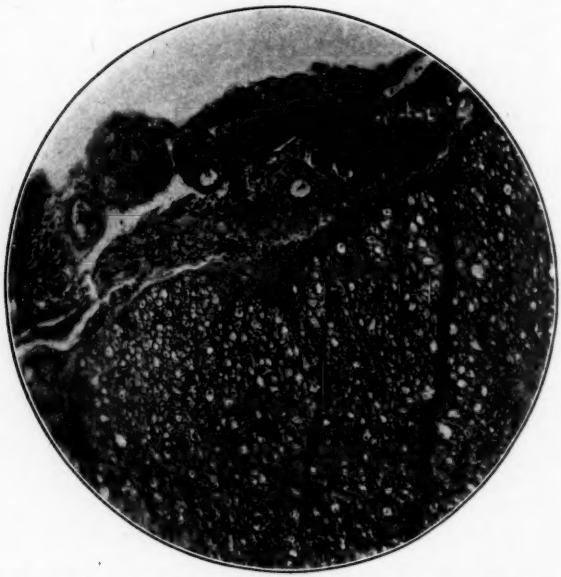

FIG. 6.—Careful examination shows well-defined patches of true proliferation in the meninges and hemorrhages.

in the lower extremity, and disorder of the function of the bladder. Practically he had no other symptoms until the acute septic condition occurred. Had he been examined for the first time on his return to the hospital and without the history of previous paraplegia, we should have had no hesitation in making the diagnosis of syphilitic spinal paralysis. Moreover, had his course of improvement been unbroken by the septic condition which overtook him, there can be no doubt that he would have eventuated in a typical example of this disease. Therefore we venture to believe that the pathological findings of this case are of great importance as indicating the anatomical foundation of some cases of syphilitic spinal paralysis.

For instance, the patient whose history is summarized below is today considered a case of syphilitic spinal paralysis, and yet at the onset of his disease he presented a parallel to the case that we publish herewith. A man, who contracted syphilis when twenty-two years old, and who received mercurial treatment by the mouth for three months, was seized four years after syphilitic infection with a weakness of the lower extremities, which within twenty-four hours reached such a degree that he was unable to stand or to walk; accompanying this there was inability to void the urine, followed by incontinence, inability to control the rectal spincters, and a sensation of numbness,

FIG. 7.—Sharply defined degeneration of the columns of Goll in the cervical region and of the direct cerebellar tract.

"weight" and "dead" feeling of the lower extremities. He gradually recovered strength in his lower extremities, and a few weeks afterward he went to the neurological department of the Post-Graduate Medical School, where he was seen by one of us.

He was given active mercurial treatment and soon disappeared from our observation. The day following the reading of this paper, that is, twelve years after his first examination, he reappeared in the Clinic. In this interval he had been earning a livelihood as a canvasser. His complaint was of stiffness and weakness of the lower extremities, hesitation in starting the urinary stream, occasional hurried action of the vesical sphincter, impotency, and paresthesia

of the lower extremities. The physical signs were exaggeration of all of the jerks of the lower extremities, the Babinski toe phenomenon, and a slight amount of rigidity, with marked spasticity of walking and a slight but distinct hypesthesia. In other words, he is today an absolutely typical case of Erb's syphilitic spinal paralysis.

We repeat, therefore, that the case whose anatomical details are herewith published may be looked upon as an important contribution not only to the morbid anatomy of the disease but as an indication of how the disease may develop and comport itself as well.

From a pathological standpoint the example that we present is one of syphilitic myelitis. The changes in the bloodvessels, the true inflammatory softening, the patches of productive meningitis, slight though they be, are in support of this statement. Contrasted with the case of Wimmer and with the case of Long and Wiki and of Williamson, it is parallel in many respects both clinically and pathologically. It is quite conceivable that if the patient had lived, inflammatory and necrotic foci would have been replaced by connective tissue, and that if his spinal cord had been examined ten years later, the secondary degeneration and connective tissue replacement would have been the striking pathological features, while there would scarcely be any evidence of inflammatory disease. The examination of an example of the disease in its incipiency, such as this one, shows that some cases, at least, of syphilitic spinal paralysis are dependent upon syphilitic myelitis.

The manner in which the diseased bloodvessels radiate to the inflammatory foci is very suggestive of the pathogenesis of the disease, particularly as we know that the spirochetes travel along the sheaths of the bloodvessels. The destruction of the tissue of the spinal cord substance may be due to the direct activity of the spirochetes themselves, to toxins which they manufacture, or it may be due to the œdema consequent upon the inflammatory reaction. It is much more legitimate to conceive this to be the cause of the destruction than to make it dependent upon disease of the bloodvessels. In some places the softening has no apparent relationship to bloodvessels whatsoever; there is simply an œdema, a round cell proliferation, and a resulting fragility of the focus.

The complete and clean-cut degeneration of Goll's column in the upper dorsal and the cervical regions is undoubtedly secondary degeneration, and we are inclined to the opinion that the ascending and descending degeneration in Gowers' tracts, and the descending degeneration in the pyramidal tracts is likewise secondary.

HABITUAL OR RECURRENT ANTERIOR DISLOCATION OF THE SHOULDER.[1]

I. ETIOLOGY AND PATHOLOGY.

By T. Turner Thomas, M.D.,

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Probably few major operations in surgery have more brilliant records, and at the same time have received less attention from the profession, than that of capsulorrhaphy for habitual or recurrent dislocation of the shoulder. The number of recorded cases in which this operation has been performed, while small, is still large enough, and the good results positive enough, to establish its claim for recognition. The greatest attention has been given to it by the French and the Germans, though few of their surgeons have operated on more than one case, Francke[2] and Perthes[3] alone having operated on four. Souchon,[4] in his elaborate paper on irreducible dislocations of the shoulder, seemed to include the habitual or recurrent as a variety of the irreducible, and drew no distinction between the two forms, so that one is led by his paper to infer that the frequently unfavorable results of operation for the irreducible apply also to the habitual or recurrent. The pathology and the result of operative treatment, however, differ substantially.

Frequency. Statistics on the frequency of this condition could not be found. It has been said that an example of it exists in every village. I have been surprised by the frequency with which I have met with cases since operating on my patient. From casual references to the subject, and through the efforts of the patient operated on, I have seen or heard of 16 cases. The frequency of the recurrences varies widely in different cases. In one of Francke's there were five recurrences in four months; in another they occurred "recently" almost daily; and in still another, several times a day "finally." The great majority of the cases recorded in the literature recurred much less frequently. The longest intervals between the recurrences with which I am familiar occurred in one of the 16 unrecorded cases. This patient received his first dislocation in a fall from an old-fashioned, high-wheeled bicycle, about twenty years ago. With fair regularity, the recurrences have occurred at about three-year intervals. In another of these 16 cases the luxations took place about every two years, for twenty years, and then ceased to occur. There is still, however, a "snap"

[1] Read in abstract at a meeting of the Philadelphia County Medical Society, October 14, 1908.

[2] Deut. Ztschr. f. Chir., 1898, xlviii, 399.

[3] Ibid., 1906, lxxxv, 199.

[4] Trans. Amer. Surg. Assoc., 1897.

or "give" on extreme abduction of the arm, indicating, probably, that the dislocation would occur from much less violence than would be necessary to produce it in the normal shoulder. Occupation has much to do with the frequency of the recurrences. The patient mentioned, for instance, has been well able to train himself to the necessity of keeping his arm at his side, or of preventing the dangerous movements; so that the infrequency of the recurrences and the final disappearance of them, in his case, may be explained in this way. Most patients cannot do this. Their occupations or inclinations compel them to abduct the arm and to take the chances of a recurrence. With each repeated luxation the facility for repetition is usually increased, sometimes to such an extent that only a turn in bed is necessary, as in Joseph Muller's[5] and Hildebrand's[6] cases.

Impairment of Function. The degree of disability varies considerably, but is always a serious matter to the patient, and may sometimes prevent him from following his usual occupation. Thus, Krumm's,[7] Samosch's,[8] and one of Perthes' patients were unable to work, and another of Perthes' patients was almost incapacitated. Generally, after a few recurrences, the patient is afraid to place his arm in the abducted position, and when he makes the attempt to do so he is seized with a "panicky" feeling from fear of a recurrence. Sometimes a mere rotatory movement at the shoulder will throw it out, so that the resulting limitation of motion greatly incapacitates the patient. I know of one patient who binds his arm to the side of the body during sleep to prevent a recurrence.

Generally, in the interval between the recurrences, there is little or no disability in the shoulder beyond the limitation of movement due to fear of recurrence; but in some cases, immediately following each luxation and its reduction, there is distressing pain in the shoulder, and in some, swelling. When, in such a case, the recurrences are frequent, the patient may be in continual distress. In one of Perthes' cases, although the last luxation had occurred two months before, the patient was still carrying the arm in a sling, to avoid painful movements.

Causes. The causes which Stimson[9] discusses at some length, and seems to regard as important, may be grouped as follows: Defects in the head of the humerus; defects in the glenoid cavity; rupture of the outward rotators of the humerus or avulsion of the greater tuberosity; detachment of the capsule from the anterior margin of the glenoid cavity; and enlargement of the joint from relaxation of the capsule. Bardenheuer,[10] in his work on injuries of the upper extremity, rejects as a cause the healing of a capsular tear

[5] Münch. med. Woch., 1900, xlvii, 1380.
[6] Archiv. f. klin. Chir., 1902, lxvi, 360.
[7] Münch. med. Woch., 1899, 986.
[8] Beiträg. z. klin. Chir., 1896, 803.
[9] Fractures and Dislocations, 1907.
[10] Die Verletzung der oberen Extremitäten, Stuttg., 1886, pt. ii, p. 414.

enlarging the joint, suggested by Malgaigne. The communication of the joint with the subscapular bursa through an abnormally wide opening (Roser), he apparently accepts. The relaxation of the capsule, he thinks, is dependent upon the distention due to the accompanying hydrops. Diminution of the size of the humeral head from the associated defects, he says, has the same effect as enlargement of the capsule. While there is a considerable difference of opinion as to the relative importance of these causes, and although a few of them have been rather generally excluded, there seems an inclination to discuss them, indicating that we are still in the stage of uncertainty as to their significance. The unity of the profession as to the pathology of this condition has not yet advanced far beyond the position of Bardenheuer, who suggested that the causes are very little understood.

In the presence of so much confusion, there is a refreshing simplicity in the proposition of Ricard,[11] that the only important factor is the enlargement of the capsule. He did nothing to prove this, except to present the argument that in the interval between the recurrences, often prolonged, the joint can functionate in a manner altogether normal. This, he says, shows that the lesion is limited to the capsule, and is a simple diaresis with integrity of the other articular and periarticular structures, skeleton, and musculature. In his two operations, he determined the integrity of the intra-articular structures by palpation through the unopened capsule. He appeared to ignore the findings in the autopsy and postoperative specimens, which had been reported before the appearance of his paper. Interesting changes have undoubtedly occurred in the head of the humerus and the glenoid cavity, and much has been written of the tearing of the muscles inserted into the greater tuberosity or of the tuberosity itself. They continue to receive much attention as causal factors from writers on this subject, although the increasing number of operations tends to strengthen the Ricard idea. One of the chief results of my study of this condition is the firm conviction that the essential lesion is the tear in the capsule which permitted the first escape of the head of the humerus from the glenoid cavity, and that the other lesions, which have been variously interpreted, are of secondary importance.

Pathology. No one, so far as I have been able to learn, has yet made use of experimental luxations of the shoulder on the cadaver, in elucidating the recorded findings at autopsy and operation. Allis' classical work on dislocations of the hip was based almost exclusively upon his experimental investigations on the cadaver. What occurred in the cadaver he assumed occurred in life, and I am not familiar with any serious objection raised against his conclusions. During the past nine years I have produced on the

[11] Verneuil, Bull. d. l'Acad. d. med., 1894, xxxi, 330.

cadaver many anterior dislocations of the shoulder for demonstration in the lectures of J. William White, professor of surgery, and G. G. Davis, associate professor of applied anatomy, in the University of Pennsylvania, and have dissected a considerable number of such specimens. Some of these dissected specimens have left in position only the torn ligaments and the bones, others the muscles in addition, and some the important bloodvessels and nerves. These specimens were used in conjunction with other dislocations on the cadaver in which no dissection was made. During these years I have come to recognize as constant and most important in the specimens the tear in the anterior and lower portion of the capsule, through which the head of the humerus had escaped (Figs. 1 and 2). The dislocations were invariably produced by abducting the arm at the shoulder until a tearing was felt or heard,

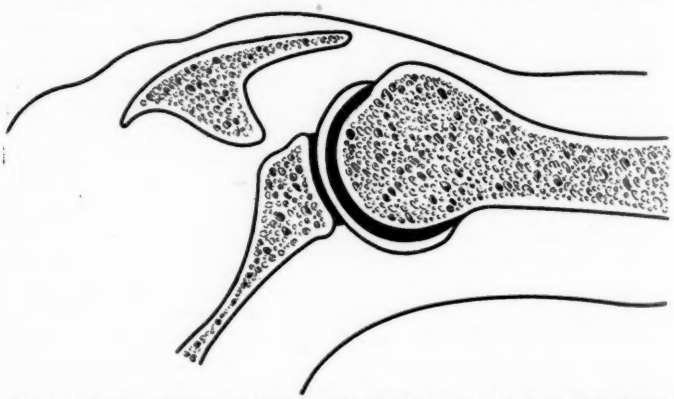

FIG. 1.—Diagrammatic section through the shoulder-joint, with the arm at right angles to the body, showing normal relations of the head of the humerus to the glenoid cavity and capsule.

when the head was forced under the coracoid process with the aid of rotation, internal and external. It is assumed here that it is generally accepted that anterior luxations of the shoulder are essentially due to abduction at the shoulder. Dissection, so far as my memory supports me, invariably showed a tear of the anterior and lower part of the capsule, and almost, if not always, from the glenoid margin, only the posterior part of the capsule remaining intact. We know that the effectiveness of Kocher's method of reduction in anterior luxations of the shoulder depends upon the untorn posterior portion of the capsule, just as Bigelow's circumduction method at the hip-joint depends upon the intact anterior part of the capsule at that joint. Allis has pointed out that the capsule in hip luxations, sometimes tears from the acetabular margin, sometimes obliquely from the acetabular to the femoral attach-

ment, and at other times from the femoral attachment. He has also shown that in the last instance the head of the femur during reduction of the luxation may push before it into the acetabulum the torn portion of the capsule now hanging from the posterior acetabular margin. I have never seen a tear of the shoulder capsule at its humeral attachment, nor one passing obliquely from the glenoid to the humeral attachment, although, because of a lack of interest in this particular point, such might have occurred and have been overlooked or not exposed. Joessel[12] reported a case of recent luxation of the shoulder which came to autopsy, showing such a tear, which he made use of in his efforts to prove that the important tear in the capsule in all the recurrent cases is at the attachment of the supraspinatus and infraspinatus muscles to the greater tuberosity. I recall one cadaver specimen in which, in addition to the usual ante-

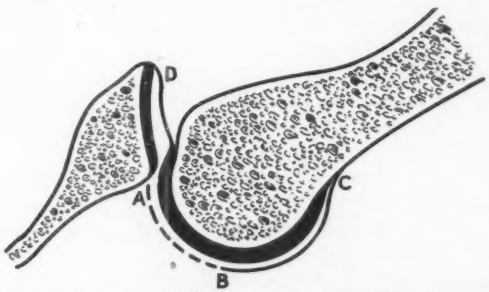

FIG. 2.—Diagrammatic section through a recurrent, anterior dislocated shoulder, with the arm in marked abduction. *B*, edge of the rent in the capsule, torn from *A*, the margin of the glucoid cavity; *C*, normal attachment of the capsule to the anterior part of the anatomical neck of the humerus; *D*, posterior untorn portion of the capsule; *A*, *B*, *C*, hernial pouch of the capsule resulting from the luxated position of the head of the humerus; *A*, *B*, new or cicatricial portion of the capsule; *B*, *C*, old or original portion of the capsule.

rior capsule tear, there was an opening into the joint posteriorly, due to the tearing off of the greater tuberosity by the attached supraspinatus and infraspinatus and the underlying capsule. In connection with the earlier specimens, such a condition did not particularly interest me, but so far as I can recall, this was the only one in which such a tearing off of bone was observed. A tearing of the muscles themselves cannot be recalled. Since beginning this study five cadaver specimens of anterior shoulder luxations have been examined, and in none of them was there any tear of the capsule, except the usual anterior one, which, in all of these, occurred from the anterior and lower glenoid margin and extended from the origin of the long head of the biceps above, to, or beyond that of the long head of the triceps below. The only exception to this in my experience was found in the case operated upon, in which it was seen clearly

[12] Deut. Ztschr. f. Chir., 1880, 167.

during the operation that the tear of the capsule occurred not at the glenoid margin, but about a half inch below and parallel to it. It extended from the level of the long head of the biceps above to at least that of the long head of the triceps below. The evidence of a tearing of the capsule from the glenoid margin in the collected cases is scanty, but it was found in some. Indeed, some surgeons, for instance, Cramer[13] and Küster,[14] could find no evidence of any tear of the capsule. This is, probably, present in every complete luxation at the time of the accident. It always occurred in the cadaver, that is, in every specimen examined. One would expect that a piece of bone would be broken off at times from the glenoid margin and that the glenoid cartilage, which deepens the cup, would be more or less disturbed. This is due either to the striking of the humeral head against the glenoid edge, or the pull of the capsular ligament. In Kraske's[15] case there was a piece of bone torn from the glenoid margin, so that the fossa was narrower below than above. It is normally wider below. Popke[16] said that the capsule was torn away from the deformed glenoid cavity in his case. In Broca[17] and Hartmann's specimen the capsule was torn away from the anterior glenoid margin, but it still remained continuous with the periosteum of the front of the neck of the scapula, which was lifted from the bone, forming a pocket into which the head of the humerus escaped. Schüller[18] noted that the anterior glenoid margin was torn off. Below it was jagged, suggesting a fracture there. He refers to experimental luxations in which such fractures were not infrequent. In Sick's[19] case the glenoid cartilage was destroyed from the lower end of the cavity up to the insertion of the long head of the biceps. The bone in this area was smooth and sclerotic and its cartilaginous margin jagged. Wiesinger[20] said that there was no tear of the capsule in his case, but a separation of it from the margin of the glenoid cavity, below and internally, through which the head had escaped. Francke found in one of his cases a piece of bone, which he said was probably torn from the glenoid cavity, although the corresponding place could not be exposed through the small incision. Hildebrand, in both of his cases, found a marked defect in the anterior margin of the glenoid cavity, and a corresponding movable piece of bone.

After reduction of a recent anterior dislocation of the shoulder, the arm is usually bound to the side of the body. This position will approximate the torn margins of the capsule or the upper torn margin to the corresponding border of the glenoid cavity, from which it was torn, and if sufficient time is permitted in this position

[13] Berl. klin. Woch., 1882, xix, 21.
[14] Verhandl. d. Deut. Gesellsch. f. Chir., 1882, xi, 112.
[15] Ibid., p. 113.
[16] Zentralbl. f. Chir., 1883, x, 28.
[17] Bull. d. l. soc. Anat. d. Par., 1890, lxv, 422.
[18] Berl. med. Woch., 1890, xxvii, 760.
[19] Jahrb. d. Hamburgischen Stattskrankanstalten, 1891–1982, iii, 374.
[20] Deut. med. Woch., 1895, v, 116.

the gap will be closed by cicatricial tissue. If, however, the pain largely disappears in a few days, as Stimson says it may do, and the joint can be freely used, the patient may, as did mine, go about without a dressing and in a short time use the arm almost as freely as before the luxation. Of course, under these circumstances the gap is not firmly closed when movements are begun; and when the arm is raised to the abducted position, a contraction of the adductor muscles of the shoulder may pull the head into the dislocated position. It is well known that progressive recurrences are usually more and more easily produced and reduced. The new space formed by the dislocated head (Fig. 2, *A B*) tends to become permanent and then becomes lined by a smooth surface, which has been observed on examination of the internal surface. This results probably from an extension of the normal endothelium from the margins of the original tear, and is reinforced externally by connective tissue, probably from a combination of the surrounding fascia and cicatricial tissue. It has been described as being of varying thickness, usually, as in my case, being thin and weak. Küster spoke of it as being thickened by cicatricial tissue. The enlargement of the capsule, which was noted as being present in practically all the cases, is due to the cicatricial new portion added to the old capsule, and is not the result of relaxation of the capsule, as has been suggested so often. Bardenheuer and others accounted for it by assuming a distention of the capsule from the accompanying inflammatory hydrops. It is surely not a gradual enlargement, but the space necessary for the dislocated head is just as large at the time of the accident as during any of the later recurrences. In my case the two margins of the original capsule tear were found and sutured together, and when this was done the original capsule seemed to be reformed without enlargement. The statements made by various writers to the effect that no tear of the capsule was found, that the capsule or joint was enlarged, or the capsule dilated or relaxed, are thus readily explained. The new, cicatricial portion of the capsule formed about the new space made by the dislocated head was not distinguished by the various operators from the old or original portion, because of the limited exposure in the deep wound.

Having stated briefly my explanation of the pathology of habitual or recurrent dislocations of the shoulder, I will consider the most important of the opposing theories with the object of showing how much they depend upon fact and how much upon misinterpretation.

Defects in the Head of the Humerus. Very striking defects have been observed, sometimes as much as a third of the surface of the head having disappeared. They are of importance chiefly because they have been made the basis of resection of the head in a considerable number of cases. A head diminished in size was considered to have very much the same causal effect as an enlarged capsule,

and the principle of the operation was that if there was no head there could be no luxation. It was always followed by a cessation of the recurrences. A very striking feature of these defects was that they were always found in the same portion of the head, the posterior and outer part of the articular surface rather below the greater tuberosity, frequently bordering on the anatomical neck. A few writers, like Perthes and Joessel, reported them as existing at the site of the greater tuberosity, but the other reported specimens (autopsy or resected) do not show the tuberosity involved. If the greater tuberosity is not involved, then the defect cannot be the result of a tearing off of the insertion of the supraspinatus and infraspinatus muscles, which is an important point. They are usually near the tuberosity, but not of it. In addition to being in the same part of the head, they are frequently of the same shape, boat-shaped, wedge-shaped, etc. As to the size of the defect, Broca and Hartmann's specimen stands probably alone. The head in this specimen looked like an orange with a quarter segment removed, which involved the whole posterior part of the head. Usually the defect is shallow, frequently only the cartilage being taken off. It was also noted frequently that in the luxated position the defect in the head rested on the anterior margin of the glenoid cavity. The area of the defect was usually deprived of cartilage, although occasionally microscopic examination showed small cartilaginous spots in it. It was usually covered by sclerotic, smooth bone, sometimes in part by a thin fibrous tissue. Sometimes there were joint bodies present made up of bone and cartilage, free or attached, and in rare instances, as in one of Francke's cases, the body fitted exactly into the defect in the head, showing that the defect and body resulted from a breaking off of a piece of the head. In other cases no such joint body could be found, and in these the defect has been explained variously: by some, as the result of the wearing effect from pressure of the head against the anterior margin of the glenoid cavity; by others, as the result of a fracture, the fragment having been absorbed.

Although pressure of the glenoid margin may have a wearing effect on the posterior surface of the head, as suggested by Löbker,[21] it seems evident to me that these defects have their origin in the first luxation. While it is generally recognized that the luxation is the result, chiefly, of abduction, there is no doubt that the head goes out through the rent in the capsule with considerable violence, due to the force of the fall on the shoulder, elbow, or hand, which is probably the usual accident producing the first luxation. As the head is being jammed forward into the luxated position it is the posterior part of the cartilaginous surface of the head which strikes against the anterior glenoid margin. In an autopsy specimen of a recent luxation, Sick found a defect of the

[21] Verhandl. d. Deut. Gesellsch. f. Chir., 1888, xv, 210.

head, 3.5 cm. by 3.5 cm., at the usual site. The degree and nature of the injury to this portion of the head will evidently depend upon the force acting and the directness with which it is met and resisted by the glenoid margin. The result may be a fracture of the head, a scraping off of its surface, or a dent in it. In the majority of the cases the head probably escapes uninjured, at least in many operations a defect was searched for and was not found. It is possible that in many of these it was so slight as to be overlooked. During the production of the luxation the corresponding glenoid margin may be partly ground off or the glenoid cartilage overlying the circumference of the fossa may be torn or crushed. While in a number of specimens it was noted that the defect in the head rested on the anterior margin of the glenoid cavity, in only two was it shown that there was a close fitting of the one into the other, proving conclusively a wearing effect. In Broca and Hartmann's specimen the marked defect in the head fitted closely over the anterior margin of the glenoid fossa, which showed no appreciable loss of substance. In Löbker's specimen the defect in the head, in a similar manner, in the luxated position, fitted over a prominent border separating the larger inner half from the smaller outer half of the glenoid cavity. These surfaces formed an angle, the inner articulating surface sloping inward and forward from the outer. In both these cases it is seen clearly that a wearing effect had been produced by the pressure of the two opposing bony surfaces. Broca and Hartmann's specimen was obtained from a patient who was admitted to the hospital unconscious and died almost immediately. He presented all the signs of an antero-internal dislocation of the right shoulder, upon which no attempt had been made at reduction. During the dissection there was a sudden jump, and the luxation was reduced. It was noted that this was clearly not a recent luxation. With no history of the case, there could have been no knowledge of the frequency of the recurrences and the period during which each recurrence had persisted, so that there might have been a more or less permanent subluxated position, when the long-continued pressure of the opposing surfaces would account for the marked wearing of one on the other.

Löbker argued from his case a like condition and cause for all other reported cases showing defects of the humeral head. Of this specimen Stimson says: "The symptoms presented by Löbker's case during life are not given, but it does not seem possible that they could have been, at least at the last, such as are found in habitual dislocation, for that is characterized by complete restoration of form in the intervals between the recurrences, while in this case the subluxation must have been persistent." Löbker, in explaining the formation of these defects in the head of the humerus and the glenoid cavity (his was the only reported specimen I found that showed such a deformation of the glenoid), says: "After

a large number of recurrences and long years of existence of the trouble, a reduction of the head into the glenoid cavity is no longer possible." This statement is not supported by other writers. The chief characteristic of recurrent luxations, as shown by the other reported cases, is that in the intervals between the recurrences the joint is, apparently, perfectly normal, except for the tendency to reluxation. Löbker, evidently, had no history of recurrent luxations in his case, the specimen having been obtained from a cadaver in the course on operative surgery, and it is likewise evident that he did not succeed in reducing the luxation on the cadaver. The only conclusion that I can reach, after studying Löbker's report, is that he had a specimen of a permanent subluxation of the shoulder, and that it has received unmerited attention from a large number of the writers on this subject. It has only served to confuse our conception of this condition. R. W. Smith[22] reported the autopsy findings in a double subcoracoid dislocation, with an excellent illustration. The history of this condition during life was not known, but the description of the shoulder-joint, in my opinion, shows the case to be exactly the same type as Löbker's, yet Smith concluded that his was the result of a congenital malformation of the joint. From a study of the other cases in which defects occurred, one is led to the conclusion that they are usually small and unimportant, and that even when large enough to involve the posterior third of the head, they do not require resection. They are, probably, just as frequent, relatively, in those dislocations which never recur as in those in which the recurrences become habitual.

Tearing of the External Rotators of the Humerus. In my opinion, the only serious obstacle to the elucidation of the pathology of recurrent, anterior, shoulder dislocations is to be found in the work of Joessel, who reported the results of examination of nine specimens of this condition. Four of these were obtained from the bodies of three persons who were known to have had recurrent dislocations during life, one having had it on both sides of the body. The other five specimens were found in the dissecting room, during two winter sessions, from an examination of 200 cadavers, and of these five, it was not said that two occurred in one body. The inference to be drawn from this is, I would take it, that we may expect one recurrent luxation of the shoulder in about every forty persons, a percentage so high as to cast some doubt on the accuracy of Joessel's observations. Only four of these five dissecting room specimens are described with any detail. They are all said to answer in every essential to those which were taken from the bodies of the patients known to have recurrent luxations during life. In not one, however, was it shown that the luxation could be pro-

[22] Fractures and Dislocations, 1847.

duced. In discussing Joessel's specimens I will confine myself to the latter, as they will serve every essential purpose. Reports of only four other authentic autopsy specimens were found in the literature, and one of these (Löbker) I have already excluded, for reasons given. The other three were reported by Perrier,[23] Broca, and Hartmann, and Sick. Joessel concluded from his study of his own specimens that the predisposition to recurrence of the anterior shoulder dislocations was due to the tearing of the supraspinatus and infraspinatus muscles with the underlying capsule from the humerus; but that it was also favored by the probably constantly occurring enlargement of the capsule. He held that when the supraspinatus and infraspinatus muscles tear away from the greater tuberosity, the head of the humerus will no longer be limited in its excursion to within the borders of the shallow and narrow glenoid cavity, so that from the contraction of the adductor muscles of the shoulder it is readily made to pass over the glenoid margin into the luxated position. He found a tearing of these muscles in every specimen, and also an enlargement of the capsule. In the first specimen the joint was filled with mercury, and found to contain 90 c.c. Normally, he says, the capsule contains only 28 c.c. With the supraspinatus and infraspinatus muscles the underlying capsule was assumed to have been torn from its humeral attachment, and the head of the humerus was said to have escaped through this tear. Perthes, while admitting the success of the operation, based upon the simple enlargement of the capsule, claimed a greater importance for the tearing away of the muscles inserted into the greater tuberosity. Much of his unusually elaborate paper is taken up by his efforts to support Joessel's position.

I appreciate fully the difficulty of disputing successfully Joessel's observations on his own specimens, especially after nearly thirty years of more or less generous and general approval by the profession. It is not, however, to what Joessel saw and described that I would take exception, but to his interpretation of his findings. It is not a simple matter to interpret the significance of the changes in such specimens long after the period of repair of a traumatic lesion. The chief point to decide is whether we have to deal with a tearing of the capsule from its posterior and superior humeral attachment, or a tearing of the capsule at its antero-inferior glenoid attachment, for the escape of the head into the anterior dislocated position. I believe that the pathology is a simple one, and that if this were recognized, most of these sufferers would accept operation and by it be permanently and almost, if not perfectly, cured.

The one sign of a tear in the capsule occurring in the original luxation, that could be detected years later, would be found in the

[23] Bull. et. mem. d. l. soc. d. chir. d. Par., 1878, iv, 112.

cicatricial tissue and adhesions existing in the immediate neighborhood of that tear. To distinguish by such changes between a tear at the anterior and inferior glenoid margin and one at the posterior and superior humeral attachment about two inches away is not without some difficulty. The only evidence of the tearing of the supraspinatus and infraspinatus muscle from the greater tuberosity, found in every specimen by Joessel, that he gives, depends chiefly upon statements to that effect; that is, he says that they were torn, the torn ends were retracted and attached to the capsule more posteriorly under the acromion. The illustration which he offers is diagrammatic and unsatisfactory, and does not increase the force of his description. Some of the specimens showed evidence, he says, of fatty degeneration, and in two at the sites of the insertions of the muscles into the greater tuberosity the bone was rough. In not one was a fragment of bone found in the retracted torn ends of the muscles. In every instance the capsule could be traced upward from its humeral attachment to the under surface of the acromion. It passed, he says, from its usual attachment about the greater tuberosity, but reached only the anterior border of the acromion. The under surface of the acromion formed a part of the upper wall of the joint. Close in front of the acromion was a protrusion of the capsule, intimately attached to the deeper part of the deltoid muscle, and separated from the rest of the capsule by a shallow groove. These statements are essentially common to all of the specimens. If they prove anything, it is that the cicatricial changes resulting from the healing in of the tear in the capsule corresponded in position to that of the upper part of a tear at the glenoid margin, and not to a tear at the greater tuberosity. Distances here are small, especially with reference to the location of the more or less diffuse adhesions of the capsule, but so far as they can be located from Joessel's description, they do not support his conclusion. The fact that he could not trace the capsule to the glenoid margin is strong evidence that the original tear was in this region. If the external rotators and the underlying capsule had torn from the greater tuberosity and the head had continued to escape through this gap at each recurrence, then the cicatrix with its assocated adhesions to the overlying deltoid muscle should have been found at the greater tuberosity. Joessel shows clearly that they were not. Perthes says that no writer other than Joessel has observed that the acromion formed the upper part of the joint in this condition.

As for the atrophy and fatty degeneration of the supraspinatus and infraspinatus muscles, it is not easy to determine these changes in such small muscles. If one were satisfied as to their presence, he would still be unable to say that they were not due to the years of limitation of function occasioned by the fear of recurrence. Under these circumstances, these signs cannot be of much value in deciding the existence of an old tear in the muscles. The statements concern-

ing the retraction of the torn ends and their attachment to the capsule more posteriorly cannot so easily be disputed; yet, in my opinion, they are not convincing. The majority of writers, in describing their operations, speak of dividing the deltoid muscle or separating it from the pectoralis major to the capsule. They say nothing of the rotator muscles, the subscapularis, supraspinatus, infraspinatus, and teres minor, which cover the capsule almost entirely. A few did refer to them with more or less detail. The explanation for this failure to observe these muscles is probably to be found in

FIG. 3.—The left shoulder in normal position, with the arm at the side of the body (cadaver specimen). *A*, clavicle; *B*, spine of scapula; *C*, acromion process; *D*, triceps muscle; *E*, coracoid process; *F*, greater tuberosity, with the common tendon of the external rotator muscles inserting into it. The muscular portions can be seen terminating, some distance posterior to the tendinous insertion; *G*, lower cut end of the deltoid muscle.

the fact that at a variable distance from their insertions into the humerus, usually about an inch (Fig. 3), they end in thin, flat tendons, which spread out and fuse with one another. This is particularly true of those which insert into the greater tuberosity, the supraspinatus, infraspinatus, and teres minor. In this tendinous portion they are intimately blended with the capsule underneath, look very much like capsule, and could here be easily mistaken for it, with a limited exposure in a small wound. The muscular bellies end some distance posterior to the humeral attachment. To ex-

amine the capsule, Joessel dissected these muscles from their scapular attachments outward. It is easily conceivable that he concluded that the anterior attachments to the capsule, normally some distance posterior to their humeral insertions, were the retracted torn ends of the muscles. His illustration, in my opinion, rather indicates a normal condition than a tear of the tendons. In none of the other three recorded autopsy specimens was such a tearing of these muscles found. Perrier's specimen was recorded in a paper which appeared two years before Joessel's paper, and made no reference to such a lesion. In Sick's specimen, the supraspinatus, infraspinatus, and teres minor muscles were said to be attached to the humerus in their normal places and were not atrophied; while Broca and Hartmann said that they did not accept Joessel's conclusion on this point, because their specimen did not show such a tearing of the muscles.

Perthes, who is the most recent writer to support strongly Joessel's findings, found a tearing of these muscles in two cases operated on —a tearing of the capsule from the anterior glenoid margin in one, and an enlargement of the capsule in a fourth operation. Of the first two, he said that there was no adhesion of the capsule to the deltoid in the region of the greater tuberosity. The blunt separation of the deltoid from the capsule was easily carried out. The head of the humerus, he says, covered by capsule and muscles, was easily exposed and showed nothing abnormal. A cicatrix here would not have permitted such an easy exposure. It was only after opening the joint and palpating its inner surface with the finger that he found the evidence upon which he based his statement that the supraspinatus and infraspinatus muscles were torn from the greater tuberosity. This evidence consisted of a rough area of bone, bared of cartilage, about the size of a mark, in the position of the greater tuberosity. He says that, more than that, a part of the capsule could be lifted from the humerus by the finger in the joint space. Defects of the cartilage in immediate proximity to the greater tuberosity have been demonstrated by the recorded autopsy and resection specimens, but in all of them it was evident that the tuberosity itself was not involved; so that they could not be due to the tearing away of the muscles inserted into the tuberosity. They were probably due to the striking of the posterior surface of the head against the anterior glenoid margin in the luxated position. The lifting of the joint capsule from the humerus by the finger in the joint space has no significance, as this can be done normally. Although Perthes implied that the separation from the bone could be carried farther downward than normal, there is nothing else in his report to prove it, and any statement so indefinite should not be accepted as proof. While I would not deny the possibility of such a tearing of these muscles in recurrent dislocations of the shoulder, the evidence of it which I have found in the literature is far from convincing.

Joessel makes the statement that "the upper capsular tear was abundantly large enough to allow the head to escape and to take its place under the coracoid process." In this I see the strongest evidence in Joessel's whole paper, that his interpretation of his specimens was basically wrong. He says that in every one of them the tear of the muscles involved only the supraspinatus and infraspinatus muscles, the corresponding portion of the underlying capsule being torn also. Through such an opening it is impossible for the head to escape, as anyone may see by making it on the cadaver, by knife or scissors. It should be borne in mind, in estimating the value of Perthes' support of Joessel's position, that the latter found this tear in every one of his specimens, and implied that it was to be expected in every recurrent luxation—that it was the essential lesion. Perthes found it in only two of his four cases. His two others, he says, represented two other groups of cases—those due to tearing of the capsule from the anterior glenoid margin and those due to enlargement of the capsule. As already stated, I regard these two as one and the same, the enlargement of the capsule resulting from a capsule tear usually at the anterior edge of the glenoid fossa. It may tear from its humeral attachment in a recent luxation sufficiently to permit the head to escape, as the capsule of the hip may tear from its femoral attachment (Allis). Such a case would probably never result in a recurrent luxation, because in the reduction, as Allis has shown in the case of the hip, the torn capsule would be pushed by the head into the glenoid cavity, and would then intervene between the head and the glenoid fossa, making the reduction incomplete and probably inducing a limitation of motion that would prevent future luxations.

If, as Joessel maintained, the supraspinatus, infraspinatus, and teres minor muscles so limit the excursion of the head of the humerus to within the borders of the glenoid cavity that the latter cannot be overstepped, then it would be impossible to dislocate the head into the complete subcoracoid position on the cadaver without tearing these muscles. Yet I have often done this and have never seen the muscles torn (Fig. 4). Joessel must have had some experience with experimental luxations, for he says that the external rotator muscles are not torn in them. He does not attempt to explain away this argument against his conclusions. The one cadaver specimen, in which, as already stated, the greater tuberosity was torn off, merely shows that the head had been pushed farther forward than was necessary for the production of the luxation, and the fact that this fracture has been shown to have occurred in recent luxations, does not prove that it is necessary for a recurrence. I have not found that an autopsy or operation has ever shown it to have occurred in a recurrent luxation, and there is reason to believe that a fracture here will prevent the development of recurrences. It would, in all probability, induce considerable pain on

movement in the shoulder for some weeks after the accident, so that the patient would be compelled to keep the arm at the side of the body long enough to permit complete and close healing of the tear in the capsule, when recurrences from the usual slight force would not take place. Stimson says: "Clinically it is known that after a few days or weeks marked by gradually diminishing tenderness and swelling, the joint can be freely used without pain." It is obvious that when the joint can be freely used in a few days without pain, there is no fracture. My patient experienced his

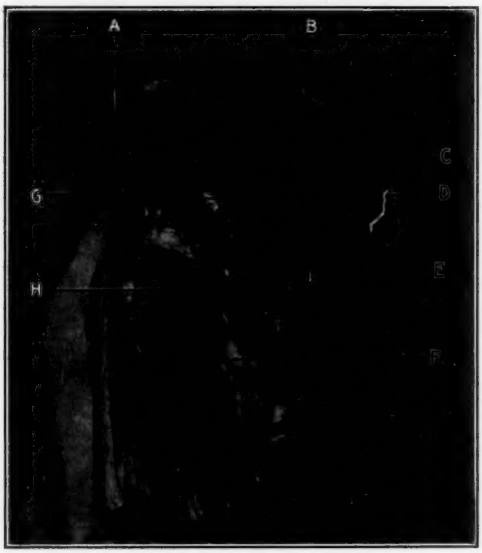

FIG. 4.—Postero-external view of the left shoulder, with complete subcoracoid dislocation. The external rotators not torn and are not under severe tension. *A*, clavicle; *B*, spine of scapula; *C*, acromion process; *D*, supraspinatus muscle; *E*, infraspinatus, *F*, teres minor; *G*, coracoid process; *H*, greater tuberosity.

first luxation in a football game. He did not confine the arm with a bandage, and five days later played in a similar game, when he had his second luxation. This fact would argue against a fracture in his case.

Neither Joessel nor any other surgeon, so far as I have been able to learn, has claimed that the tear in the capsule necessary for a recurrent luxation differs from that necessary for a recent, although Stimson seems to imply it. Joessel, because he believed that a tear of the supraspinatus and infraspinatus and the underlying capsule was necessary for every anterior luxation of the shoulder, advised that in the treatment of the first luxation the arm should be

kept in the horizontal position in order to approximate the margins of the muscle and capsule tear, and to obtain close union during the period of repair. This would be logical if his pathology were right. There are few physicians today who would not advise that the arm be kept at the side of the body, that is, in adduction, during the healing period; and everyone who does employ this treatment rejects Joessel's findings in his specimens of recurrent anterior luxations of the shoulder. The following, from his book on fractures and dislocations (1907), would indicate that Stimson still accepts Joessel's teaching: "This tendency (to habitual dislocation) has generally been attributed, although without anatomical proof, to laxity of the capsule, itself the consequence of imperfect repair of the rent made in it at the time of the dislocation; but the recent researches of Joessel show, for the forward dislocation, that the enlargement of the capsule observed in such cases sometimes takes place at the upper portion in consequence of the rupture of the capsule at the same level and the creation of a free communication between its cavity and that of the subcoracoid (subacromial) bursa." Stimson, however, says of recent subcoracoid dislocations of the shoulder that the capsule is torn at the lower and inner portion. According to Joessel, the tear is in the upper and outer portion. Stimson dresses the arm at the side of the body with inward rotation, to approximate the margins of the rent during the period of repair. Joessel, as we have seen, advises abduction of the arm at the shoulder to obtain the same result. That Stimson really does not accept Joessel's pathology of recurrent dislocation of the shoulder is shown best by the fact that in two cases he followed the Ricard principle, of only shortening the capsule anteriorly, with good results, and recommends this operation as the best.

While most of the surgeons who, in recording cases, have taken up the pathology of this subject, have given more or less credit to Joessel's findings, I have found only two who have applied the principle involved, in their operations. Francke, in one of his four cases, excised an oval piece at the site of what he regarded a muscle tear (supraspinatus and infraspinatus), and brought the edges together, thus shortening the capsule and bringing the torn edges of the muscles nearer to their normal insertions. There was no recurrence four months later. No later observation was reported and no reference to function, so that we cannot fairly compare this case with any of those in which the anterior relaxed portion of the capsule was shortened without suture of the muscles. Perthes, in two of his cases, brought what he supposed were the torn ends of the same muscles as far forward as possible toward their normal insertions. He drove iron staples into the humerus, toward which he drew the muscle ends. He likewise shortened the relaxed capsule by reef sutures of silk. It was, in all probability, this feature of the operation which produced the good results, and they were

surely not better than those obtained from simply shortening the capsule. In his first case, two years after the operation, and in his second case, three months after operation, the affected arm could be raised vertically to an angle of 160 degrees. My patient, three months after operation, could elevate the arm straight upward, or to an angle of 180 degrees. Probably all that was accomplished by suturing the muscles was to shorten them slightly and thus to impair their function. Probably, and fortunately, the sutures cut through, and the new adhesions were broken up by the active and passive movements of the after-treatment in the weeks following the operation, so that the effect of the sutures was largely overcome. When a partial dilatation of the capsule was noted, it was practically always of the anterior, internal, or axillary portion, and is readily explained by an original tear of that portion.

As a result of my study of the subject, I am led to the simple conclusion that the usual anterior, habitual, or recurrent dislocation of the shoulder is due to a traumatic, cicatricial, anterior, hernial pouch of the capsule, and that the object of the operation should be to obliterate the hernial protrusion. It would be a simple matter to remove at the same time any free or attached joint body that may be present. Joessel's teaching is based upon a misinterpretation of his specimens, and should be set aside.

The treatment of this condition will be taken up in a subsequent paper.

THE CLINICAL FORMS OF PYELONEPHRITIS.[1]

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It is difficult to find a correct name for the condition to be described in this paper. The term pyelonephritis is, without doubt, the proper one from a pathological standpoint for those cases which I shall speak of as the urogenous type, that is, those in which the infection extends upward from the bladder to the pelvis of the kidney. In the other variety, that is, the hematogenous type, in which the infection is carried to the kidney through the blood stream, the pelvis of the kidney is but little affected until late in the disease, so that the pathologist would term these cases septic nephritis. The clinical picture, however, of both types is so similar that it seems best for the present to retain the term pyelonephritis

[1] Read at a meeting of the Mississippi Valley Medical Association, October 14, 1908.

to include both modes of infection. Henry Morris[3] calls the condition to be described "acute interstitial nephritis," but this, and the term "infective nephritis," lead to so much confusion with well recognized clinical forms of nephritis that it is well to discontinue their use in this connection.

Pyelitis and pyonephrosis have been well recognized conditions for many years. The intermediate pathological condition, pyelonephritis, in which the kidney is the seat of suppuration in its earlier stages, has not received the consideration which it deserves. It has become an absolute necessity at the present time for us to be able to recognize such a pyelonephritis clinically in order to be able to treat it properly.

ETIOLOGY. The causes are most conveniently divided according to the chief modes of entry of the infection: the urogenous or ascending, and the hematogenous or descending.

1. *Urogenous Type.* (*a*) Cystitis is one of the most frequent sources of infection. The presence of cystitis alone would not be sufficient to explain the spread of infection along the ureter to the kidney. We know that normally the back flow of urine from the bladder into the ureter is prevented by a number of factors, chief among which is the sphincteric closure of the ureter. Israel and others have shown that in cases of enlarged prostate and stricture of the urethra, there is a diminished action of this sphincter, and the orifice becomes patulous, thus allowing the urine to back up into the ureter. Any condition, such as cystitis, which increases the normal intravesical pressure, will produce the same effect. Again, a loss of contractile power on the part of the bladder walls, such as occurs after enlarged prostate or injuries and diseases of the spinal cord, will produce the same result. (*b*) Any obstruction to the outflow of urine from the kidney, whether it be in the pelvis of the kidney, ureter, bladder, or urethra, may be a factor. This urogenous cause includes calculi, strictures, tumors pressing upon the ureter, enlarged prostate, and kinks of the ureter in floating kidney. (*c*) Fistulæ between the ureter and other hollow viscera, such as the rectum, which will permit of the entrance of organisms into the ureter, may also serve as a cause.

2. *Hematogenous Type.* (*a*) Primary. This includes the cases in which the foci of infection in one or both kidneys are the only demonstrable ones. This is the form to which attention has of recent years been directed by Brewer, Cabot, and others. In some of the cases the condition has followed a trauma, and Brewer has demonstrated experimentally that an injury favors localization of the organisms; but so many cases occur without an injury that the latter cannot be regarded as a vital factor. (*b*) Secondary. This embraces a large class of cases in which the renal infection is preceded,

[3] Surgical Diseases of the Kidney.

(1) by one of the acute infectious diseases, such as pneumonia, typhoid fever, erysipelas, or diphtheria, etc.; (2) by a focus of suppuration elsewhere in the body, such as a furuncle, carbuncle, phlegmon, osteomyelitis, etc.; or (3) by some form of bowel infection. The latter is the most frequent source of infection in the cases of pyelonephritis in children, as shown by Heubner, Baginsky, and Abt.

PATHOLOGY. The organisms most frequently found in both the urogenous and the hematogenous types are colon bacilli, staphylococci, streptococci, gonococci, and proteus vulgaris. It is generally believed that the gonococcus simply prepares the soil for the other varieties by producing a preceding cystitis, ureteritis, or pyelitis.

The colon bacillus and the ordinary pus organisms undoubtedly play the chief role in the majority of cases, whether of hematogenous or urogenous origin. The pathological changes vary according to the source of infection and the virulence of the organism. In the urogenous or ascending type the changes characteristic of an acute or chronic pyelitis precede the renal changes. The latter differ from those found in the hematogenous type by the fact that the first evidence of infection of the renal parenchyma in the urogenous type is in the form of yellowish streaks, extending from the papillæ into the medulla and cortex. In addition, one finds multiple miliary abscesses scattered through the cortex. A characteristic feature of these pyelonephritic abscesses is their frequent arrangement in groups, surrounded by zones of intense hyperemia, best seen on the surface of the kidney. Each miliary abscess is surrounded by a dark zone of intense hyperemia. Whether the abscesses occur singly or in groups, they usually project beyond the level of the surrounding cortex and become adherent to the capsule. If there is much obstruction in the pelvis, ureter, bladder, or urethra, the back pressure greatly favors the destruction of the kidney, and all stages of the process can be found from the small abscesses to transformation of the kidney into a pus sac (pyonephrosis).

The hematogenous form differs somewhat in its pathology from the urogenous. There are no preceding changes in the pelvis of the kidney, especially no yellowish streaks of purulent softening radiating from the papillæ toward the cortex. The hematogenous type may appear in such a hyperacute form that only congestion and turbidity of the kidney parenchyma are found, death taking place in a few hours after the onset of symptoms. In the less acute form the changes are somewhat similar to those found in the urogenous type. Throughout the parenchyma, numerous foci of infection are found. These show all of the stages of suppuration from leukocytic infiltration to necrosis of the tissues and pus formation. As in the urogenous type, the most characteristic evidence of infection is the presence of a large number of miliary abscesses on the surface of the kidney. There is, however, not as much tendency to the group-

ing of the abscesses as in the urogenous type. In some of the hematogenous cases a single or several large abscesses have been found, instead of numerous small ones. Goodhart, in 1868, and Weir, in 1894, showed that the urogenous type very frequently involved only one kidney. Prior to that time the old term "surgical kidney" was usually interpreted as a bilateral infection of both kidneys following a cystitis, enlarged prostate, calculi, etc. Attention has of recent years been directed to the fact that in many of the cases of hematogenous infection the disease is quite often unilateral. This latter fact is very important in the diagnosis and treatment of the condition.

Symptoms and Diagnosis. There is scarcely any other renal disease which presents such a variety of clinical pictures as pyelonephritis. A description of one group of symptoms would not answer for every case. It is, therefore, most convenient to divide the clinical forms of pyelonephritis, first, according to the source of infection, that is, whether it has reached the kidney through the blood stream (hematogenous) or through the medium of the ureter (urogenous). This division will suffice for quite a number of cases in which the previous history and the examination of the urine will show whether or not the bladder has been the source of infection. There are, however, an equally large number of cases in which the source of infection is not quite clear, and yet the clinical picture of a renal infection is quite evident. It is so difficult to classify the latter class of cases that it has always seemed advisable to me to place them in separate groups. The cases to which I refer are those occurring (*a*) in children, and (*b*) in the puerperium and during pregnancy. Clinically, then, such a division would be as follows:

1. Hematogenous type: (*a*) Hyperacute; (*b*) acute; (*c*) subacute.
2. Urogenous type: (*a*) Recurrent febrile; (*b*) continuous febrile (chronic urosepsis).
3. Pyelonephritis of children.
4. Pyelonephritis of pregnancy.
5. Pyelonephritis of the puerperium.

1. Hematogenous Clinical Varieties. For many years the view was universally held that renal infections (not including calculous pyonephrosis or renal tuberculosis) were almost always bilateral and the result of a preceding cystitis. For this reason the now obsolete term of "surgical kidney" was employed to include every clinical variety of the disease. We now know that the source of infection can be recognized from the symptoms and examination alone as being hematogenous in origin, and that in a very large proportion of cases the infection involves only one kidney, and is thus amenable to surgical intervention. Israel[3] was one of the first to call attention to hematogenous renal infection as a clinical entity.

[3] Surgery of the Kidney, 1901.

Since that time George E. Brewer, Farrar Cobb, I, and others have described the clinical picture of these cases of acute hematogenous infections of the kidney. The clinical types of the latter are:

(*a*) *The Hyperacute Hematogenous Form.* The clinical course is usually so rapid and the symptoms of a most virulent infection so marked that any symptoms pointing to the kidney as the source of infection are obscured by the general sepsis. The onset of symptoms is very sudden, usually in persons who have previously enjoyed good health. There is great prostration, high fever (to 106° F.), rapid pulse, marked leukocytosis, vomiting, and often general muscular pains. In some cases there is severe abdominal pain, muscular rigidity, and tenderness over the costovertebral angle. When localizing symptoms such as those just mentioned are absent, and the examination of the urine is negative, the diagnosis must be one of exclusion. The conditions which must be considered in the differential diagnosis are severe influenza, acute abdominal affections, such as perforation of gastric and duodenal ulcers, appendicitis, gallstones, mixed infection in tuberculous kidneys, malaria, and typhoid fever. Unless the appendix lies behind the cecum, or high up near the liver, the differentiation from appendicitis is not difficult. In the latter disease there is more muscular rigidity and tenderness, and pain is a far more prominent symptom. In the case of the retrocecal appendix there is tenderness and pain in the iliocostal space, but there is apt to be more muscular rigidity and spontaneous pain than in acute renal infection. In many cases examination of the urine will be of great aid in distinguishing the two conditions.

In acute cholecystitis the pain is more superficial and nearer the costal arch. Muscular rigidity is most marked in the right hypochondrium, and tenderness on palpation more marked there than in the iliocostal angle. The differentiation of the other conditions named usually presents no difficulties.

(*b*) *Acute or Intermediary Hematogenous Form* (Brewer). The clinical pictures in the less acute or intermediary (Brewer) hematogenous forms of infection differ but little from that of the acute urogenous type. The chief difference lies in the fact that the symptoms in the hematogenous type begin either after one of the acute infectious diseases or soon after a septic focus, such as a carbuncle, etc., has appeared in some other part of the body. In the urogenous form there is often the history of a preceding cystitis, or there are evidences of the presence of obstruction in the urinary tract, either in the form of a valve, stricture, or stone in the ureter, or of an enlarged prostate, or calculi obstructing the urethra.

In these acute cases there is the history of a sudden onset of fever, often accompanied by chills. The temperature is irregular and often persistently high, and is accompanied by all the other evidences of a severe infection. Fortunately, the localizing signs both in the acute and subacute forms are more marked than in the hyper-

acute. There is usually some tenderness over the iliocostal angle, and often muscular rigidity here.

(*c*) *Subacute Hematogenous Form.* In this third form of hematogenous renal infection the principal symptoms are long-continued fever of an irregular character, accompanied by unilateral costovertebral tenderness. Urinary findings are more constant in acute and very mild forms than in the hyperacute. Pus, blood, casts, and albumin are present in the majority of cases. The diagnosis in this mild class is made by a thorough examination so as to exclude any other source of fever taken in conjunction with the costovertebral tenderness and urinary changes.

2. Urogenous Clinical Varieties: (*a*) *Recurrent Febrile Urogenous Type.* The symptoms of this form are quite similar to those of the hematogenous subacute form just described. The history differs somewhat: As in the acute hematogenous form, a patient who has had a cystitis will suddenly have one or more chills, followed by high temperature and profuse sweats. This syndrome will last several days to a week, and then the temperature will become normal or slightly above normal for a variable period. A second attack of chills, fever, and sweats will occur as unexpectedly as the first, and the same cycle will repeat itself a number of times.

The diagnosis in these cases presents no difficulties if one will examine the urine for pus, blood, and casts, which are almost invariably found. The amount of pus is always larger just after the cycle just described, since it is the retention of this pus in the kidney which causes the recurrent febrile attacks.

(*b*) *Chronic Urogenous Pyelonephritis.* In this group of cases the renal infection is always secondary to a cystitis. The latter may be a complication of a hypertrophied prostate or a stricture of the urethra. Such mechanical obstructions to the outflow of urine are not a necessary factor in the production of a cystitis which is followed by chronic infection. Cystitis may exist in both sexes, but with especial frequency in the female, without such mechanical obstruction, and remain unimproved for months and years. In this class of chronic urogenous renal infections the clinical picture is often that of a pyelitis alone. Pathologically, however, the renal parenchyma is equally as much involved. Clinically these cases present a most varied picture. In some, fever is more or less constant. The temperature ranges from 99° to 100°, rarely higher. There is an inability to gain in weight in spite of the most careful attention to diet and general hygiene. Such patients lack color, and in some the anemia is quite noticeable. A dull aching pain in the lumbar region is quite often present. The urine contains pus in variable quantities.

An effort was made by older writers to distinguish pus coming from the kidney from that due to a cystitis by the fact that in the former case (kidney) the urine was acid and epithelial cells with

small bodies and long tails were present in the urine. These epithelial cells were said to be characteristic of the renal pelvis. In cystitis, the urine was said to be alkaline, and there was an absence of the aforementioned epithelial cells. We now know that these older views as to the source of the pyuria, although true in a measure, cannot be relied upon to distinguish pus of vesical from that of renal origin. The reaction of the urine depends upon the microörganism which causes the infection. If it is due to the colon bacillus alone, as is so frequently the case, the urine is acid, whether the bladder alone or both it and the kidney are involved. If the infection is due to the urea decomposing organisms, the reaction is alkaline. In regard to the epithelium, it is a well-recognized fact that metaplasia or change in form occurs so frequently in the bladder epithelium during a cystitis that no reliance can be placed upon any particular form as distinguishing bladder epithelium from that coming from the renal pelvis. The only positive manner in which the source of a pyuria can be determined is by a careful study of the clinical symptoms taken in conjunction with the results obtained by cystoscopic examination and ureteral catheterization.

In many of these chronic urogenous pyelonephritides there are periods in which they have higher fever and other more general signs of infection, which rapidly subside as soon as the drainage from the kidney is free again. Influenza, malaria, intestinal autointoxication, rheumatism, etc., are often diagnosticated in these cases, when in reality the source of the recurrent or protracted fever, etc., is in the kidney.

3. Pyelonephrisis during Infancy and Childhood. This form only differs from those just described by the fact that it is a frequent cause of obscure fever in infants and young children, and as such deserves special mention. In a recent paper I. A. Abt has reported 22 cases of cystopyelitis, and calls attention to the presence of persistent fever in children, accompanied by rapid pulse and progressive pallor and emaciation. The fever is frequently interpreted as due to an intestinal intoxication, and unless pyelonephritis is borne in mind as one of the causes of obscure fever during early life, the diagnosis will either not be made at all or the condition will be recognized too late to be of any benefit. The mode of infection in the majority of these cases is undoubtedly through the blood stream, and the source or atrium for the organisms to enter the circulation is in the intestine. The acute febrile form with the clinical picture of a severe sepsis ushered in by chills, high fever, etc., does not occur nearly as often in children as in adults. An examination of the urine should be made in every case of continued or remittent fever in children when no other cause can be found.

4. Pyelonephritis during Pregnancy. The symptoms of renal infection occurring during pregnancy differ but little from the

description of the clinical picture in males and in the non-pregnant female. The only reason for describing it separately here is to call attention to the fact that when it does occur during pregnancy the possibility of renal infection is seldom taken into consideration in making a diagnosis of the cause of either a continuous fever or of chills and fever, which occur without warning.

A recent case of mine will serve to illustrate many of the important points: During the fifth month of her pregnancy, a patient who had previously enjoyed the best of health had a chill, followed by a temperature of 105° F., accompanied by rapid pulse, great prostration, high leukocyte count, and scanty urine containing much pus. The temperature dropped to normal within a few hours, but the same cycle of chill, fever, and sweat recurred at irregular intervals during the following four days. The urine contained a few casts and some epithelial cells, which were interpreted as of renal origin. There was not more albumin than could be accounted for by the amount of pus present. I saw the patient in consultation on the fourth day of the illness. Palpation of the abdomen failed to show any localized muscular rigidity or tenderness over the right iliac region. One could make deep pressure over all parts of the abdomen, but there was no sensitiveness except upon bimanual palpation of the right kidney, and here it was only of moderate degree. Examination of the genitalia was negative. One could thus exclude appendicitis, cholecystitis, or any form of genital infection. The possibility of such a sudden onset, during pregnancy, of chills, fever, etc., even without vesical symptoms, was recalled, and after appropriate non-operative measures were instituted, the patient made a prompt recovery. Pregnancy proceeded without further incident and ended in a normal manner.

This case illustrates one of the two clinical pictures under which renal infections can occur both during pregnancy and in the puerperium. In the second form, the disease may pursue a more chronic course. There is a general feeling of malaise, accompanied by muscular pains, fatigue, and fever of varying degree. It is the persistence of this temperature which first directs attention to this second class of cases. Bladder symptoms, as in the more acute form, may be entirely absent or very slight. In the chronic form the presence of pus in the urine is a more constant finding than in the acute form, because the infection is usually the result of the ascending type, that is, from the bladder. Tenderness over the kidney or ureter is seldom sufficiently marked in these more protracted cases to be of value in making a diagnosis. When the outflow of pus from the kidney is not obstructed there is but little fever, but this recurs promptly when there is any retention. The use of the cystoscope is seldom necessary, since the diagnosis in both of the above classes of cases can be made by excluding any other source for the fever than the kidney.

In the majority of cases the pyelonephritis is upon the right side. This is due to the fact that although both ureters are compressed by the uterus during pregnancy, the right ureter is more compressed than the left, as the result of the rotation of the uterus on its long axis from left to right and the greater frequency of positions in the right oblique diameter. The most frequent abdominal conditions which begin so acutely are cholecystitis, stones in the common bile duct, and appendicitis. These can be readily excluded by the absence of localized muscular rigidity and tenderness over the respective areas, and the fact that high fever preceded by chills is only characteristic of common duct stones.

5. Pyelonephritis in the Puerperium. This, like the similar condition in pregnancy, may begin very suddenly, without prodromal symptoms like those of cystitis. In the majority of cases the source of the infection in all probability is in the bladder. In only a small number is the infection a descending (that is, a hematogenous) one. The patient may have made an excellent recovery from the parturition with normal temperature, pulse, and lochia. Then suddenly she has a chill, followed by high fever, rapid pulse, and a marked leukocytosis. The fever either persists or recurs irregularly following a chill.

In some cases there is marked pain over the affected kidney (in the lumbar region) and along the ureter. In others there are absolutely no localizing symptoms referable to the kidney. This syndrome may occur during the first few days or several weeks after parturition has occurred. Pus in a variable quantity is usually present in the urine; and in some cases there is vesical tenesmus and pain on micturition, but these two symptoms are not constant. The diagnosis in these cases must be made by excluding any of the pelvic complications following infection during or soon after parturition. If the pelvic examination yields negative results, an infection of the kidney should always be thought of as a frequent cause of fever during the puerperium. The clinical picture just described, of a sudden onset with chill, fever, etc., does not fit all cases. In some, the only symptom to attract the physician's attention is rise in temperature, especially toward evening, for which no cause can be found in the genitalia Palpation of the kidney in both of the above types often reveals considerable tenderness, especially on bimanual palpation. The organism which causes the pyelonephritis is the colon bacillus, and the starting point is without doubt an intestinal infection, especially in those cases in which any instrumental infection of the bladder can be excluded.

THE OPERATIVE TREATMENT OF DEFLECTION OF THE NASAL SEPTUM.

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The consideration of the proper operative treatment for the restoration of the deflected nasal septum is only secondary in importance to that still important question as to the justification of the operation in any individual case presenting itself for consideration. Since attention was first called to the deflection of the septum, and the possible influence that bending of the septum—obstructing one or both of the nasal chambers more or less completely—may play in the causation of certain pathological changes in the nasal cavities, the ingenuity of rhinologists has been taxed to overcome this deflection with the restoration of the septum to a normal vertical plane.

The nasal septum is a thin plate of cartilage and bone covered with mucoperichondrium and mucoperiosteum, and separates the nose into two nasal cavities. The mucoperichondrium and mucoperiosteum are very loosely attached to the bone and periosteum, except at sutural borders and where from traumatism it has formed adhesion. This structure is composed of the quadrangular cartilage, the vomer, and the perpendicular plate of the ethmoid. It should be placed in a vertical plane in the nose to be in an absolutely normal position. Through the action of various agents the septum becomes subject to alteration from the normal plane; when this change is so excessive as to obstruct one or both nasal cavities, or bring it into contact, more or less constant, with one or more of the turbinates, the condition becomes pathological. The changes in the septum may be such that there is a simple bending of the septum without any thickening; at other times the deflection is attended with increase in its later dimensions, which latter may assume the form of thickening at the point of greatest bending or the formation of a distinct ridge or spur.

Etiology. The etiological factors which produce deviation of the septum may be classified under four headings: Developmental, obstructive, traumatic, and diseases of a general or local nature.

Developmental. This factor no doubt plays quite an important role in the causation of deflection of the septum. The septal constituent parts not ossifying with equal degree of rapidity, and being in excess of the normal space allotted to them, cause, naturally, a bending of the later ossifying bone or the least resisting constituent of the group making up the septum.

Obstructive. Conditions causing obstruction to the air current through the nasal cavities impair the development of these chambers

and the constituent bones that enter into their formation. At the same time there is frequently an increase in the arch of the hard palate, leaving, consequently, less vertical space in the nose within which the septum must form.

Traumatic. Direct violence of great force is frequently sufficient to fracture the bones of the septum, causing displacement of them with resulting deflection; or by separating the quadrangular cartilage from its attachment to the vomer and ethmoid it causes luxation of the latter, with resulting deflection of the structure. Mild blows, frequently repeated, received throughout childhood, cause occasionally displacement, but most frequently thickening along the sutural borders.

Diseases of a General or Local Nature. It is stated that syphilis and rachitis are factors in developing deflections, but wherein they can be causative agents I have never been able to see. Local inflammatory disturbances producing adhesions in the young and newgrowths are infrequent causative agents.

Symptoms. A deflected septum in itself is not a pathological condition. The deflection becomes pathological only when it causes impairment of the function of the nose either through partial or complete arrest of the air current through one or both of the nasal cavities, or when through pressure upon the turbinate bodies, intermittent or continuous, the cause interferes with the performance of the normal functions of these bodies; or, when through one or both of these disturbing causes, a certain train of symptoms is produced which are usually designated as reflex.

It is not always possible to come to an immediate conclusion wherein and how far a deflected septum is giving rise to symptoms. The compensatory function of all organs of the body is so great that often gross pathological lesions apparently cause no marked alteration of function. This compensatory function in the nose is well marked, so that ofttimes marked deflection causing complete obstruction of one nasal chamber apparently gives rise to no inconvenience to the patient. At other times, when the objective evidence is not very pronounced, most frequently when the whole plane of the septum is bent toward one cavity, narrowing but not markedly obstructing the affected side, the disturbance to nasal breathing and the general health of the patient is very marked. At times it is very simple to observe the necessity for operative intervention. The obstruction is patent and the subject evidence, as described by the patient, is positive. Those cases with moderate deflections and marked vasomotor disturbances also tax the skill of the diagnostician, as regards the relation of cause and effect. Oftentimes it becomes necessary to examine a patient several times, at intervals, under different conditions, to come to a safe and sure opinion. Indeed, at times, when a septum has been operated upon after one had somewhat

of a struggle to come to the operative opinion, one will find, well back beyond the deflection, prominent ridges or spurs, which projected into the turbinate, and which were entirely hid from view by the deflected portion of the septum. At the present day, when operative intervention about the septum has become so common, it is well that one consider the case quite thoroughly as to the extent of the deflection, as to its impairment of respiration, how far it influences pathological changes within the nasal cavity, and to what extent the reflex symptoms present in the individual case can be associated with the deflected septum.

The only condition which may stimulate a deflected septum and would possibly deceive one who is competent and skilful enough to perform this operation is a gumma of the septum. I recently saw a gumma involving the cartilaginous septum in the upper two-thirds of its extent on one side, which had been brought to me for operation. On the deflected side there were the evidences of deflection, as shown by the rounded gumma; on the other corresponding side of the septum there was a flat, plane surface. An indefinite history of syphilis was obtained. Three months later I saw this patient with a perfectly flat septum on both sides.

TREATMENT. In the early period of nasal surgery it was recognized that the deflected septum played an important role in the impairment of function and the causation of certain pathological changes, so that rhinologists early sought methods to overcome this condition. The early attempts at correction were vain, and more failures by far were the result of operative treatment than improvements—very few good results. Various forms of penetrating punches, as well as stellated cutting forceps, were used at that time, with solid metallic or ivory splints to retain the so-called straightened septum. Then came the operation of Roberts, the cutting of the septum and pinning the septum into its new position. We next find the varying types of cutting and crushing operation, which had for their purpose the destruction of the resiliency of the septum, thus restoring it to the vertical plane, where it was retained by splints in its new position until the attempt had proved a success or failure. The cases that resulted favorably through this method of operation were those that permitted the riding over of the fractured fragments. The redundant septum thereby reduced itself so that it could more nearly approach the normal type. Under this class would be included the Asch, Roe, and other operations too numerous to mention.

The operations for deflection of the septum, done under the plan suggested by Asch and Roe, yielded often excellent results in the hands of the projectors of these operations; nevertheless, in the hands of others who essayed this form of operation, failures and untoward results were as frequent as successes. It has been my experience to re-operate upon a number of cases subjected to this

form of operation, in which nothing seemed to have been gained by the former operative procedure. Later came special modifications of these operations, all being an improvement on the previous more or less blind, crushing method, and each, in certain types of deflection, being attended with most excellent results. Under such groupings may be classified the Kyle, Watson, Gibbs, and others following out the same general technique. All the above-mentioned operations have in common the cutting, or cutting, sawing, and fracturing of the displaced septum, thus removing redundant tissue or permitting the over-riding of the fragments and reducing thereby the redundancy of tissue, and leaving, after healing, a replaced, firm septum in its normal anatomical integrity.

During the greater portion of this period there had been gradually developing a type of operation which had for its purpose the removal of the deflected portions of the septum, whether cartilaginous or osseous, or both, through a submucous method, leaving a straight, although a partially flaccid septum. Through the efforts of Myles, Killian, Hajek, Freer, and others, this method has been perfected in the last five years, and, as perfected, has been accepted by the greater portion of the rhinological world as the operation for the correction of misplaced septa. Those operators who had gained the greater portion of success in their operative cases through the employment of one or more of the previously designated operations were rather loath to accord fellowship to the new—to them untried—method of operating.

The objections raised against the new operation were: (*a*) The possibility of lowering the plane of the dorsum of the nose; (*b*) the great possibility of causing perforations through the new method of operating; (*c*) the question as to the advisability of replacing a normal osseocartilaginous septum with a semiflaccid septum; and (*d*) the recognized difficulty of the technique, and the time consumed in the operation. Previous to 1904 I had done all my operative work upon the septum by a method that had given me a more than fair degree of successful issue. For several years failure in operative treatment, with restoration of the septum to a normal or nearly normal plane, was rather infrequent by my method. I was, therefore, uncertain as to the advisability of relinquishing my previous method for the new doubtful one. My operative procedure had for its purpose the removal of a narrow, linear wedge-shaped segment of bone from the osseous septum at the juncture of the vomer with the floor of the nose, through the use of a Fetterolf saw; the separation through sawing, or partially sawing, and then fracturing the nasal spine; cutting through the whole anterior border of the quadrangular cartilage just behind the columna, continuing this incision along the superior border to the degree necessary; the operation being completed by fracturing the anterior extremities of the vomer and ethmoid, when found necessary.

Always after this method of procedure the septum would fall into a normal vertical plane.

The test of the probable success of any of these forms of operative procedure is the fact that the septum will assume the normal vertical plane after the operation is done—retaining apparatus only being necessary to prevent displacement. In this operation, as well as all other operations done upon the septum, the individual case and its requirements should be the controlling factor which should govern the operation, that is, as to the character of the incision, the length of the incision, the amount of bone to be excised, and other details; one detail, however, should never be forgotten, and that is, sawing through and loosening the nasal spine. The retaining apparatus employed in my cases is the Kyle aluminum splint, which is worn continuously for about seven days. This operative procedure presents certain features that were original creations of mine. The fracture of the spine had been suggested and acted upon by Harrison Allen, who rightly considered it essential to the success of the operation. Allen's method of attacking the spine was by an incision made through the gingivolabial border of the upper lip. I attack the spine through the floor of the nose by means of a saw, either sawing through it completely, or partially, and then fracturing it by the use of forceps. The continuous wearing of the retaining apparatus for a week or ten days was first suggested to me. After demonstrating to my satisfaction the advisability of this method over the old procedure of frequent removal and introduction, I presented it to the profession in a paper read before the meeting of the American Laryngological, Rhinological, and Otological Society, held in Pittsburg on May 11 and 12, 1898.

Although my results had been unusually good through the former method of operation, there were yet a sufficient number of unfavorable results to wish for some operative procedure that would, with the execution of proper skill, give uniformly perfect results. As the literature seemed to bear testimony to uniform results following the submucous method, I determined to direct my attention to it.

I will briefly describe the submucous method for those not versed with its technique. The operation is most frequently done under cocaine anesthesia, either through the laying of pledgets of cotton saturated with a 10 per cent. solution or the rubbing in of the crystal of cocaine, or through the hypodermic injection of a solution of cocaine beneath the mucous membrane. The mucous membrane is also rendered ischemic through the use of suprarenal or adrenalin. After the anesthesia and ischemia are complete, a vertical incision is made through the mucous membrane and the perichondrium of the septum in that nasal cavity into which the bending of the septum presents itself. I find it preferable to incise into the cartilage in the primary incision, as well as to use the index finger in the other nasal cavity as a guide and safeguard against carrying the

incision too far. Some operators have a rule to make the primary incision in that nasal cavity in which the convexity of the deflection presents itself; other operators always from the left nasal cavity, whether it presents the concavity or convexity of the deflection. It is more convenient to operate in the left nasal cavity, as one must be expert with the use of the left hand to operate in the right nasal cavity. There is no doubt that it is preferable to operate in that side which presents the convexity.

Many, following the teaching of Killian, make a vertical incision from the dorsum, about 0.5 cm. posterior to the columna, extending to, if not including, the floor of the nose; others make the incision slightly curvilinear, but follow the same outline. Others, following the teaching of Freer, make the incision in the form of an inverted capital L over the point of greatest projection. In my early work, I followed the Killian incision, but latterly I have made the incision quite well forward, leaving only a narrow strip of cartilage between the line of incision and the columna. This has the especial advantage in that, in case a puncture is made through the perichondrium and mucous membrane when incising the cartilage, one may make another cartilaginous incision a millimeter or more farther back, and from that point raise the mucous membrane and perichondrium on the opposite side. Indeed, the deflection may be so placed as to render it necessary to make the incision directly posterior to the columna. I have never attempted the Freer flap. From the manner in which I have seen flaps retract when broken at their lower angle, and the absolute inability to draw them down so that they in any way come over the surface of the opposite flap, I should judge that Freer's flap must leave a large surface to cover by granulation.

The next step in the operation is the raising of the mucoperichondrium. The starting of the perichondrium is really the crucial point of the operation, for, as this is well done, so is the operation a success or a failure. A half sharp elevator is used for the beginning of the work. Care, patience, and the employment of plenty of time is requisite in this procedure. It is usually best to commence at the upper end of the incision, for here the soft structures are free from irritation, and will consequently separate with greater facility. One should gradually insinuate the half sharp separator between the soft tissues and the cartilage along the posterior border of the incision until he finds it raising the perichondrium slightly, then gradually work downward, producing the same degree of separation until the floor of the nose is reached. If the primary incision, at the lower point, or at any point, is not through the perichondrium, one is very apt to tear the flap at the point of non-separation. After separation is made throughout the whole length of the incision and backward for about 0.5 cm. with the half sharp separator, the final separation is rapidly finished with the employment of the full,

rounded separator, with the execution of proper care over sutural borders, over angles and points of adhesion. The mucoperichondrium should be separated thoroughly over the hard parts as far as the removal of cartilage and bone may be expected to extend.

The second important step is the incision through the cartilage made through the original wound and parallel to it without injury to the mucoperichondrium of the opposite side. This is one of the most important steps of the operation, and must be performed with exquisite sensitiveness of feeling and nicety of skill so as not to make a puncture through the perichondrium and mucosa. A puncture is almost certain to be attended with a subsequent perforation, however minute it may be. Very many methods have been suggested to obviate the use of the knife in this procedure, but in my experience, if extreme care is exercised, no other expedient is necessary. If such a misfortune is met with, it is wise to abandon the incision made through the cartilage and make another parallel incision through the cartilage a millimeter back of the original incision, and from this raise the perichondrium of the opposite side over the same area, and with the same care and delicacy with which it has been carried out on the other side. Always be careful to see that the mucoperichondrium on both sides has been well separated down to the floor of the nasal chambers. The third step is the removal of the deflected cartilage and bone between the two flaps which have been formed. Many remove the cartilage and bone between the flaps without any means of protection to the flaps. I consider this method unnecessary and unsafe. I always use the Killian long speculum to widen out the flaps and protect them from injury. The Killian, or some similar form of speculum, should always be used during this stage of the operation. The final procedure is the bringing of the flaps together and holding them in position by a packing of gauze. The gauze is usually removed in twenty-four hours, when union is found to be complete. The foregoing is practically the usual operative procedure. I have varied this slightly in my technique.

From observations which I have made in the clinics and private work of some of the most dexterous and rapid workers in this operative technique, and from my own experience, I early became convinced that cocaine anesthesia was not sufficient to relieve the patient of pain attendant upon its performance; also, that a suffering patient, with loss of fortitude, did not aid in the successful issue of such a delicate operation. In my early work I operated under cocaine anesthesia. In the early stage of the operation, under the use of cocaine, I found the anesthesia all that could be desired, but, as the operation was often prolonged, and the most trying part of the procedure came at the last, I found that most of my patients suffered quite a little pain and shock, and several of them became so demoralized as to render the completion of the operation extremely difficult. I therefore turned to general anesthesia, at first only operating upon

those under this method who I feared would not last out under local anesthesia. I found this method so satisfactory that, during the last two years, I have done all my septum operations under general anesthesia. By this method the patient is entirely free from the possibility of pain, and the operator is saved the disquietude resulting from the anxiety and suffering of his patient. The bleeding under general anesthesia is a little greater than under local anesthesia, but I have never found it sufficient to delay or annoy me during the progress of the operation. I have also found it wise to study carefully the relation between the convex surface of the deflection and the alæ of the nose, so that the incision will be made sufficiently well forward to avoid making a failure of the operation, on account of the alæ coming in contact with that portion of the cartilage that is left.

My results by this operation have been uniformly good for the relief of stenosis, contact with the turbinates, or the relief of reflex symptoms. In none of my cases has there been any sinking of the dorsum of the nose. Where there is sufficient bony structure left above and a fair bridge of cartilage anterior to the seat of operation, I doubt if this occurrence can take place. I doubt very much if there is ever a reproduction of cartilage or bone in the area from which it is taken. The flaccid septum becomes tense and later gives a certain firmness of sensation. In several of my cases in which a large portion of the hard septum was removed, my patients have been annoyed for a month or more by the flapping of the flaccid septum. The symptom of which they complained was the frequent alternate occlusion of the nasal cavities, due to the flaccid septum coming in contact with one, then the other, outer wall of the nose. I, unfortunately, have had perforations. Most of my perforations were made during my early operative days, and no doubt were largely the result of want of knowledge of the adequate skill to obviate and overcome the various pitfalls which in this operation present themselves from the very start to the finish of the operation. I know of no operation in which an operator has to be so "keyed" up and so carefully has to scrutinize every move in the technique as is required of a surgeon in this operation. Twice I have had two fairly large perforations to develop from pin-point incisions into the opposite mucoperichondrium at the point opposite the primary incision. In both of these cases I was conscious of the wound, and, before introducing the packing, I closely scrutinized the mucous membrane, hardly being able to note the point of injury; nevertheless, the perforation resulted. In all I have had 3 per cent. of perforations. In my last series of 80 cases I have had only one perforation, and this was one of the 2 described above. Twice when operating I have recognized the fact, from a misfortune, that a perforation would in all probability result if I continued the operation by the submucous method; I therefore discontinued the procedure and resorted

to my former method. Perforations resulting after this procedure heal kindly and are not usually attended by crust formation, therefore give no annoyance to patients; in fact, patients are never conscious of their existence unless informed of it. If we could depend upon our confrères to be considerate in not notifying the patients of the existence of a perforation in their noses, they would be of no consequence, except the personal chagrin they cause the operator. During the last three years I have operated upon 190 cases of deflected septum by this method; 22 of the operations were done under local anesthesia, with 3 perforations; 168 were done under general anesthesia, with 5 perforations. During this same period I performed 20 operations by the method formerly employed by me, with only one perforation. The results gained through the two methods of operative procedure have been practically the same, except that, in the submucous operation, the septa presented cosmetically a more perfect appearance.

In sharp, angular deflections the introduction of the index finger into the cavity presenting the angle and gently but firmly forcing it over will cause it to assume a more rounded form so that the separation of the mucoperichondrium can be accomplished with more ease. Great care should also be exercised in seeing that ridges at the junction of the vomer and the maxillary crest have the mucoperichondrium raised so that they may be thoroughly removed. It is better to remove these ridges with the chisel, as through this instrument they can be expeditiously and more satisfactorily removed.

In considering the advantages which the various operations for the correction of the displaced septum offer over one another, we must not lose sight of the personal equation of the operator. Many operators can perform the older methods of straightening with a marked degree of success, and have not the patience or requisite skill to operate by the submucous method. Most operators find that experience is a great teacher in the submucous operation. Manœuvers that seemed almost impossible in the earlier operations become easy of manipulation after many have been done.

1. Conclusions. The old method gives a septum maintaining its anatomical integrity. The submucous operation gives a flaccid septum, minus certain elements formerly considered essential to its anatomical integrity. This removal of the cartilage and bone seems to have no influence upon the function of the parts, unless removed over too great area toward the dorsum of the nose.

2. The operation by the old method requires about ten minutes in its consummation; the submucous requires about one-half hour in actual operating time.

3. The old method requires the wearing of a splint for a week or ten days; the packing in the submucous operation is removed in twenty-four hours.

4. In the old method there is considerable pain and discomfort until the splint is removed; in the submucous there is no postoperative pain.

5. In the old method of operating the patient is confined to bed for several days, and is uncomfortable and feverish until the splint is removed. After the submucous operation the patient is out of bed at the end of twenty-four hours, and at the end of forty-eight hours is practically well.

6. The old method is rarely attended with perforation. I have had only two in my whole experience. Theoretically, the submucous method should be attended with like results; practically, perforations do occur. After performing one hundred and ninety operations by the submucous method and nearly twice as many by the older method, I have become firmly convinced that the submucous method offers the greatest advantages to the patient afflicted with a deflected septum, in the hands of an operator thoroughly skilled in its technique. I know of no operation wherein unskilled and inexperienced hands can do more harm than by this operation. I feel as though I am becoming a strong advocate of the submucous operation, because it gives less discomfort to the patient, it taxes the skill of the operator, and, when satisfactorily done, gives excellent results.

THE FURTHER REPORT OF A CASE OF TRACHEAL SCLEROMA.

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Two years ago I presented to the American Laryngological Association the history of a case of scleroma of the larynx.[1] Ten months later there was a recurrence of the growth and the patient presented herself hoarse and with dyspnœa very marked, and gradually becoming worse. Noting the remarkable results obtained in other cases of external rhinoscleroma by the use of the Röntgen-rays, I felt that their application direct to the part through an opening in the trachea might have a curative effect. In any event tracheotomy was becoming daily more necessary, and if benefit was not to be obtained the patient would be compelled to wear the tube, as had others before her, to prolong her life.

The patient readily consented to the operation and subsequent direct treatment by the Röntgen-rays, and on March 25, 1907, she

[1] Amer. Jour. Med. Sci., 1907, cxxxiii, 751.

was admitted to the Mt. Sinai Hospital, in the service of Dr. A. G. Gerster.

The following are the notes of the examination by myself, on March 28: In the nose half way back on the left side is a grayish-white mass which bleeds very readily when touched and is covered with thick secretion. This mass does not occlude the nose on that side. On the right side is another similar mass partially occluding the nose. In the trachea, below the cords, on a level with the cricoid cartilage, is a mass attached to the wall of the trachea and extending downward. This is an extension and continuation of a condition existing for over a year under my observation, and is a scleroma of the larynx verified by the pathologist's report. The diagnosis is increasing tracheal stenosis due to scleroma, with secondary involvement of the intranasal mucous membrane.

Physical examination on March 30 showed: A fairly well-nourished young woman; facies somewhat dyspnœic; hoarseness: respirations stridulous, difficult, gasping, and considerably prolonged in both phases; percussion signs somewhat diminished, especially posteriorly at the bases; apices free; vocal fremitus diminished; voice and whispering sounds diminished below in front and over the bases posteriorly; numerous sonorous and whistling rales over both lungs, anteriorly and posteriorly; in the axillary regions and over the bases posteriorly numerous moist rales, increased after coughing.

On March 30 Dr. A. G. Gerster did an extensive tracheotomy. The incision extended three and one-half inches from the lower edge of the thyroid cartilage to about three-quarters of an inch above the sternal notch. The isthmus of the thyroid was tied off in two places and divided between ligatures. The cricoid cartilage and tracheal rings were incised, bringing into view the tumefaction of the posterior and lateral aspects of the trachea. The newgrowth was grayish white in appearance, resembling soft mucous polyps readily breaking down, and extending one and one-half inches below the larynx. A large tube was introduced and packing about it to keep the wound open.

On April 1 the packing and tube removed. The edges of the wound were held apart by retractors, and under chloroform anesthesia there was a five-minute exposure to high-frequency current at a distance of eight inches.

April 3: has had frequent paroxysms of coughing. Whenever she sits up, is fed, dressed or in any way disturbed, these coughing spells occur, and frequently vomiting ensues. These phenomena were not present before *x*-ray treatment.

April 9, 12, 15, and 17: *X*-ray treatment by Dr. Stern, five- to six-minute exposures.

April 20: The growth has greatly decreased in size, and the trachea looks much wider. Has had no more vomiting and her

tracheal reflexes are less active. It is now possible to dress the wound and clean the tube without exciting coughing spells as formerly. *X*-rayed on the 22d and 24th.

May 10: Patient has had three or four *x*-ray exposures a week; an examination of her larynx by myself showed an absence of any tumefaction, and I recommended permanent withdrawal of the tracheotomy tube and further treatment externally. Voice now normal and no dyspnœa.

On May 11 the tube was removed; she remained until May 17 when she was discharged from the hospital cured. During her stay in the hospital her temperature never arose above 100.5°. After the first few times no chloroform was given prior to *x*-ray treatment. Urinalysis frequently made showed neither albumin nor sugar.

Examination April 12, 1908: Until the present time the patient has been in normal condition without dyspnœa, hoarseness, or cough. She has been attending to her work with regularity and only appears now at my request for examination. Her general condition is good, respiration normal, and she says that except for occasional nasal obstruction she is entirely well. On the right side of the nose about the centre of the inferior turbinate is a small pink mass which probably interferes slightly with her breathing. On the left side, on the septum at the floor, is a roundish mass about the thickness of a small hazel nut. This was removed with a cold wire snare and was about one inch in length. There was but a slight bleeding.

Examination of the larynx and trachea showed perfectly normal conditions.

More than a year has now elapsed since her operation and subsequent treatment, and no sign of recurrence is visible within the larynx or trachea, and we may justly consider her as cured of her most threatening condition.

The benefits of the *x*-ray application to external scleromas have been known, and now we can show its utility in local conditions that we are able to reach in a direct manner. Should a recurrence be present we might reasonably expect good results by external application of the *x*-rays over the cicatrix, this not presenting the same resistance as the normal tissues would; that failing it would be easy to reopen the old wound and make local application again.

I wish to express my thanks to Dr. A. G. Gerster and to Dr. Samuel Stern for their cordial coöperation and active interest in the surgical and *x*-ray treatment of the patient.

THE PRESENCE OF TUBERCLE BACILLI IN THE CIRCULATING BLOOD IN TUBERCULOSIS.[1]

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The principal reason for taking up a bacteriological study of the blood in tuberculosis was to determine whether a previous assertion, made in connection with my article on the presence of tubercle bacilli in the feces, was correct. This assertion was that, considering the entrance of the tubercle bacillus into the human economy, "the number of bacilli is not large, and they find their way to the blood and lymph stream; and during their transit some are discharged through the feces and others through the urine." Again, after examining the contents of 22 thoracic ducts from subjects dead of tuberculosis, tubercle bacilli were encountered in 72.7 per cent. This occurrence also led me to believe that all forms of tuberculosis are characterized by a bacteremia.

Some observers have been successful in obtaining cultures of the tubercle bacillus from the blood in cases of acute miliary tuberculosis, but the instances are not numerous. The studies undertaken by me were at first to prove that tubercle bacilli are present in the blood in all cases of tuberculosis, and to determine if possible the presence of organisms in the so-called mixed infections in the late stages of the disease.

Numerous clinicians have asserted that the termination of cases of pulmonary tuberculosis is accompanied by infections of the pneumococcus, Streptococcus pyogenes, staphylococci, and Bacillus pneumoniæ. My own observations from examinations of the blood previous to the present study were usually negative as regards these mixed infections, as I never obtained any other organism besides the staphylococcus in quite a number of cases, even in those of tuberculous pneumonia. The occurrence of the staphylococcus was not considered as a possible infecting organism, but was usually looked upon as a contaminating organism from the skin.

In making up my list of cases for this research I first selected those who were bedfast and who presented advanced tuberculosis or cavity formation; then cases of acute miliary tuberculosis; and lastly, instances of incipient tuberculosis.

The technique for the demonstration of tubercle bacilli in the blood was as follows: About 5 c.c. of blood was withdrawn from a vein of the arm, and this was immediately placed in an equal quantity of a

[1] Read at a meeting of the Pathological Society of Philadelphia, December 10, 1908.

2 per cent. solution of sodium citrate in normal salt solution. The mixture was well shaken and placed in the refrigerator for twenty-four hours. At the end of this time there is an abundant sediment, with the citrate solution slightly turbid. Centrifugalization immediately after mixing did not give very good results, so I determined upon letting the mixture stand for twenty-four hours. After this time I pipetted off a quantity of the sediment, made a rather thick preparation upon a new, clean glass slide, dried it upon a copper plate with moderate heat, and then placed it in distilled water until complete laking of the blood resulted. At this time only a delicate film remained. This was dried and fixed through a Bunsen flame and then stained by the usual technique for tubercle bacilli. As a rule, bacilli possessing the morphological and tinctorial properties of the tubercle bacillus could be demonstrated in the first slide thus prepared, although in several cases three slides were thoroughly searched before any bacilli were found. A negative result should not be announced until at least thirty minutes careful searching has been given to a slide.

The pipettes before being used were thoroughly washed in water, then placed in pure nitric acid, and after being used were again washed, this time in a solution of caustic soda, rinsed, and again placed in pure nitric acid until ready for use. No one pipette was used on two different cases on the same day. Lately I have sterilized the pipettes at 160° C. for one hour and then placed them in nitric acid before using. The syringes used for withdrawing the blood, as well as the needles, were boiled for about twenty minutes before and after using in a solution of caustic soda. The needles were kept in a solution of equal parts of lysol and glycerin, and just before being used were washed in sterile water. The glass piston, as well as the nozzle of the syringe, was anointed with glycerin to prevent any undue adhesion of these parts. No special preparation of the patient's arm is necessary; lysol was simply applied upon the area as Wright practises in his opsonic work.

In the first few studies undertaken 10 c.c. of blood was withdrawn, then 5 c.c., and when for some unforeseen circumstance this amount could not be obtained from the patient 2 c.c. was taken. I would recommend 5 c.c. as the proper quantity to withdraw. Intimate mixing must be made lest the blood clot, in which case the greatest difficulty arises in demonstrating the organisms.

The cases studied up to the present time number 50. Of these, 5 were diagnosticated as acute miliary tuberculosis; 2 as fibroid tuberculosis; 1 as pneumothorax; 15 as incipient tuberculosis; 23 as moderately advanced tuberculosis; and 3 as laryngeal tuberculosis. I also had the opportunity of examining the blood from the umbilical cord of a placenta delivered from a tuberculous mother.

In every case examined tubercle bacilli were found. Sometimes only a few were seen, but they were mostly in large numbers, and

clumps of 30 to 40 bacilli were not unusual, especially in the cases of acute miliary tuberculosis. In morphology most of the organisms were of normal size, though some were short and even clubbed; a few very long forms were also encountered. In one of these cases the pneumococcus was seen. In several cases clumps of tubercle bacilli could be clearly observed in some of the leukocytes, indicating phagocytosis.

In a patient in whom tubercle bacilli were found in the feces on two different occasions, and who upon physical examination did not present any signs of the disease, the organisms were found in the blood, and about 2 c.c. of the citrated sediment was inoculated intraperitoneally into a guinea-pig. This pig died in about three weeks from an internal hemorrhage (peritoneal). The lungs were studded with tubercles, four tubercles were present in the liver, and enormous enlargement of the spleen as well as enlarged mesenteric glands were found at autopsy. Spreads from the liver, spleen, kidney, heart's blood, and mesenteric gland contained tubercle bacilli, but no other organism.

In a second case, diagnosticated as acute miliary tuberculosis, 2.5 c.c. of the citrated sediment was inoculated intraperitoneally into a guinea-pig. This animal was sacrifiecd at the end of five weeks, and presented the following lesions at autopsy: Four bronchial glands ranging in size from 0.5 to 1.5 cm. in diameter, several enlarged mesenteric glands, tuberculosis of the spleen, and only a few small tubercles in the lungs. Spreads from the spleen, mesenteric glands, and blood showed a few tubercle bacilli.

Conclusions. It appears that tuberculosis in all of its forms is a bacteremia. Even in healing cases, or in those in which the disease is being arrested, tubercle bacilli are demonstrable in the blood, although usually in small numbers. In subjects dead of the disease, if histological examination of the liver, spleen, and kidney are made, one would find scattered miliary microscopic tubercles, which strengthens the above assertion. Only a small number of cases are here reported,[2] but it seems that even in this small number the constant finding, especially in cases of incipient tuberculosis, before the detection of the bacillus in the sputum, is quite a valuable procedure. Again, it appears that the theory of mixed infection late in the disease does not always hold, for the reason that if tubercle bacilli are demonstrable by this method other organisms should be just as evident, and yet in only one of the cases studied was another organism—the pneumococcus—present. This patient had a pleural effusion, but was really in the incipient stage of pulmonary tuberculosis.

[2] Up to January 15, 1909, I have studied 125 cases, and have detected the tubercle bacillus in all.

REVIEWS.

APPLIED SURGICAL ANATOMY. By GEORGE WOOLSEY, A.B., M.D., Professor of Anatomy and Clinical Surgery in Cornell University Medical College. Second edition; pp. 601; 200 illustrations. New York and Philadelphia: Lea & Febiger, 1908.

THE author's revision of this work brings it well up to date and continues it in the position it has always occupied, as one of the most practically useful of the text-books of applied anatomy. Few of them contain so many separate items of information of importance to the practitioner, and in none of them is such material better arranged or presented in a more concise or more readable form. The book is so well known and so generally appreciated that in reviewing this edition the chief interest centres in the additions or changes which, since 1902, the progress of anatomy and surgery has necessitated, or which have been suggested by the widening experience of the author. A few examples taken at random may be given: In the section on the localization of cerebral functions, it is now said: Before the investigations of Sherrington and Grünbaum this area was thought to comprise the cortex of the anterior and posterior central convolutions, bordering the fissure of Rolando, and the cortex immediately adjacent to them, especially the paracentral lobule on the mesial surface. As the result of these observations on the higher apes, confirmed, in part at least, by histological studies and the faradization of the human cortex in the course of operations, the motor area is now thought to lie entirely in front of the central fissure and to extend to the bottom of it on its anterior surface only. In the same section the existence of the heat-regulating centre in the medulla is now noted; the teaching as to the visual speech centre has been modified, and it is correctly added that as the basis of language is a series of memory pictures (1) of the sound of words, (2) of their appearance, (3) of the effort necessary to enunciate them, and (4) to write their symbols, and as these memory pictures are connected with each other and with others that make up the concept by subcortical association fibers passing between them, a lesion in any of these association tracts also leads to a defect of speech.

In the section on the neck "enlargement" of the thyroid has properly become "hypertrophy;" the function of the gland is more fully dealt with; the parathyroids and their relations are well de-

scribed and figured, and attention is called to the fact that "removal of all the parathyroids or the destruction of their blood supply causes death from acute tetany. As the loss of their blood supply is the most frequent cause of tetany, great case should be observed in thyroidectomy only to tie the arteries that actually enter the thyroid."

In the section on the thorax a few lines on operations upon the heart have been added, but, in view of the results of operative interference in cardiac wounds, the description might have been amplified with advantage. In the section on the abdomen a number of advantageous changes have been made, a few cuts added, and some conspicuously useless ones (like Merkel's "Outlines of the Abdominal Viscera") omitted. The statistics as to gastric ulcer have been corrected and brought up to date. The former edition said, "Ulcer of the duodenum, as compared with gastric ulcer, is uncommon." In the present edition it is said that "ulcer of the duodenum was formerly thought to be uncommon, but among 200 cases operated upon, 98 were of the duodenum, 87 of the stomach, and 15 of both (Mayo). They occur mainly in the first 4 cm. (one and one-half inches) of the duodenum and altogether above the bile papilla, that is, in the area where the contents are acid." The article on the pancreas has been partly rewritten and enlarged. It would bear still further enlargement. A cut of the retropancreatic lymph nodes has been inserted, but there is no reference to it in the text except a rather misleading one relative to the position of the spleen (p. 375).

In the section on the lower extremity the description of the anatomy of dislocations of the semilunar cartilages has been made both fuller and clearer. In the section on the pelvic viscera, the "anorectal line" of Testut, and Hilton's "white line"— the "anorectal groove"— are for the first time described in relation to the anal portion of the rectum, and the freedom of that portion from involvement in prolapse (on account of its firm support by the levator ani muscle) is mentioned. Houston's folds as seen through a speculum are shown in a clumsy and space-consuming plate. Two smaller but much more useful cuts (from Spalteholz), showing the relation of the sphincters and the interior of the rectum, have also been added.

The section of the spinal cord has been well revised. The italicizing throughout seems a little overdone, the desired emphasis being sometimes lost by reason of the frequency with which the eye is called upon to recognize it. The space given to the time-honored, but largely useless, "cross-sections" of limbs might have been used to better advantage. A very few typographical errors were found, *e.g.*, "scapulabis" (Plate XIV, p. 184). But, on the whole, the book has been kept up to its previous high standard of accuracy and usefulness, and is among the most satisfactory of the treatises on this subject, whether for the student, the general practitioner, or the surgeon.

J. W. W.

OXFORD MEDICAL PUBLICATIONS:

HEART DISEASE AND THORACIC ANEURYSM. By F. J. POYNTON, M.D., F.R.C.P., Assistant Physician to the University College Hospital, London.

AUSCULTATION AND PERCUSSION. By SAMUEL GEE, M.D., F.R.C.P., Consulting Physician to St. Bartholomew's Hospital, London.

MEDICAL LECTURES AND APHORISMS. By SAMUEL GEE, M.D., F.R.C.P., Consulting Physician to St. Bartholomew's Hospital, London.

CLINICAL LECTURES AND ADDRESSES ON SURGERY. By C. B. LOCKWOOD, Surgeon to St. Bartholomew's Hospital, London.

SELECTIONS FROM THE WRITINGS of SIR WILLIAM BROADBENT. Edited by WALTER BROADBENT, M.D., M.R.C.P.

GLANDULAR ENLARGEMENT AND OTHER DISEASES OF THE LYMPHATIC SYSTEM. By ARTHUR EDMUNDS, F.R.C.S., Surgeon to the Great Northern Central Hospital, London.

DIETS IN TUBERCULOSIS. By NOEL DEAN BARDSWELL, M.D., Medical Superintendent of the King Edward VII Sanatorium; and JOHN ELLIS CHAPMAN, M.R.C.S., Medical Superintendent of Coppin's Green Sanatorium.

THE TREATMENT OF DISEASE IN CHILDREN. By G. A. SUTHERLAND, M.D., F.R.C.P., Physician to the Paddington Green Children's Hospital, London.

THE SKIN AFFECTIONS OF CHILDHOOD. By H. G. ADAMSON, M.D., M.R.C.P., Physician for Diseases of the Skin to the Paddington Green Children's Hospital, London.

THE LAW IN GENERAL MEDICAL PRACTICE. By STANLEY B. ATKINSON, M.B., B.Sc., Justice of the Peace for the County of London.

LIFE INSURANCE AND GENERAL PRACTICE. By E. M. BROCKBANK, M.D., F.R.C.P., Honorary Assistant Physician to the Royal Infirmary, Manchester, England.

ROTUNDA MIDWIFERY FOR NURSES AND MIDWIVES. By G. T. WRENCH, M.D., Late Assistant Master of the Rotunda Hospital, Dublin, Ireland.

London: Henry Frowde, and Hodder & Stoughton, 1908.

THE foregoing comprise some of the excellent series of manuals recently issued from the Oxford University Press. Together they share the interest and importance that attach to monographs; they are small in compass and devoted to interesting topics, and they are written by authors well qualified by study and experience to discuss the respective subjects.

Dr. Poynton's volume consists of a clear and succinct statement of the clinical methods of investigating diseases of the heart, and of the pathology, symptoms, and treatment of the more important forms

of heart disease. Major significance and importance is attached to acute rheumatic heart disease, which is taken as a type, and from which other forms of heart disease are differentiated. This necessitates an unusual and a somewhat confusing discussion of the pathology, symptoms, and treatment; but it has the merit of emphasizing the widespread activity of the rheumatic poison—in implicating the pericardium, the myocardium, and the endocardium—and it is altogether pardonable in one whose painstaking studies of the bacterial cause of acute rheumatic fever and of rheumatic heart disease are well known and highly prized, and who has so much of value and so much that is original to relate. Especial interest centres in the chapters on myocardial and neuromuscular disorders of the heart, which are often more or less disregarded in the too common thought that heart disease and valvular disease are almost, if not quite, interchangeable terms. The discussions of treatment are rather full and very valuable: the local use of cold in acute cases is extolled; the salicyl compounds are believed to be of no value; the suggestions for the use of digitalis and strychnine are excellent. The chapters of congenital heart disease and on arterial disease and aneurysm are altogether too brief, even for a manual. The book as a whole can be warmly commended; perhaps some day the author may gratify many readers by expanding it to a more pretentious volume.

Dr. Gee's book on auscultation and percussion is now in its fifth edition—which of itself is sufficient evidence that it has for some time subserved a useful purpose. It has the special merit of being entirely personal; it relates in clear and terse language the important facts of physical diagnosis; and it avoids the distraction of extraneous discussions. It is unusually dogmatic, but its dogmatism may be excused, since the main object of stating facts is so well accomplished. Dr. Gee's *Medical Lectures and Aphorisms*, now in its third edition, comprises an unusual amount of medical wisdom and philosophy—graphically, succinctly, quaintly expressed. The general nature of the book may perhaps be best exemplificd by several quotations: "Pneumonia is not a local, but a universal disease, and the brunt of it may fall upon any part—lungs, endocardium, membranes of the brain, intestines, kidneys." "Purgatives in dropsy are not of mucb use; the practice is a survival. If we cannot act upon the kidneys, we should do nothing to add to the patient's discomfort." "In women past middle age gallstones are so common that one is not wrong to be always suspecting them." "A letter containing four pages or more, closely written, and narrating the writer's own disorders, is a sure and certain sign of hypochondria." "When we have prescribed that continual temperance in all things which is necessary to the cure of most disorders, how often do we find that our patient reckons the loss of pain to be purchased too dear by the loss of pleasure." The quaintness and

what one may perhaps speak of as the elegance of diction lend an added charm to both books, of which certain chapters should be resorted to for pleasure in a leisure hour.

Mr. Lockwood's book consists of certain clinical lectures and addresses delivered from time to time and published elsewhere. Several of the more interesting are: Clinical reasoning; the recognition and management of intestinal obstruction; the essentials of a diagnosis; carcinoma of the breast and its spread into the lymphatics; exploratory laparotomy, especially in cases of malignant disease; clinical pathology in relation to diagnosis and treatment; and salivary calculi.

Mr. Edmunds' book comprises an interesting discussion of a much neglected subject, which is considered from a practical and a surgical standpoint; its limitations have been defined by considering only those pathological conditions in which affections of the lymphatic vessels or glands constitute the essential feature of the disease, such as cellulitis, acute infections, retropharyngeal abscess, glandular fever, chronic infections (especially tuberculosis) and their surgical treatment, diseases of the thymus gland, the surgery of the thoracic duct, lymphatic insufficiency, filariasis, dilatations of the lymphatic vessels, lymphadenoma or Hodgkin's disease, lymphangio-endothelioma, lymphosarcoma, the operative treatment of secondary malignant glands, and the treatment of inoperable malignant glands. The discussions are altogether commendable; the advice in regard to treatment is especially sound and practical. The book is a real addition to medical literature.

The volume of writings of Sir William Broadbent consists of a judicious selection from among the many papers of a distinguished author. Some of these have been selected because of their historic interest, such as that on the bilateral association of nerve nuclei (so-called Broadbent hypothesis), others to illustrate the variety of the author's work in his early days of professional life, and others because they are of present-day interest. Among the last named one may mention the examination of the heart (the author's last completed paper), adherent pericardium, pneumonia, interlobar empyema, acute dilatation of the stomach, some affections of speech, and syphilitic affections of the nervous system.

Bardswell and Chapman's *Diets in Tuberculosis* comprises the results of many years' study and investigation of diet and dietetics in general, but especially in regard to tuberculosis and the nourishing of the poor. They discuss the comparative economy of various foodstuffs, the economics of diets, and certain observations on the treatment of tuberculous patients with meat-free diet. They give the diet lists of several sanatoriums, certain practical directions for out-patients in regard to buying and cooking diets, and a dietary, costing sixteen cents a day in England, that has been found adequate for the treatment of pulmonary tuberculosis. The book is an

interesting and valuable addition to the already voluminous literature on tuberculosis.

The general scope of the other books, those by Drs. Sutherland, Adamson, Atkinson, Brockbank, and Wrench, is well exemplified in their respective titles. The discussions are in keeping with the excellence of the volumes of the series already issued. A. K.

HYDROTHERAPY. By SIMON BARUCH, M.D., Professor of Hydrotherapy in Columbia University (College of Physicians and Surgeons), New York; Medical Director of the Hydriatic Department of the Riverside Association. Third edition; pp. 544; 74 illustrations. New York: William Wood & Company, 1908.

IN the third edition of his excellent text-book Dr. Baruch takes a very broad view of his subject, including even the antiseptic effects of hot water in surgery.

The first section is devoted to the physiological action of water. The various experiments cited are more interesting than decisive, as may be learned from Dr. Baruch's conclusion, "that, despite some differences of opinion and deductions, the changes produced by the thermic and mechanical stimulation arising from hydriatic procedures are very intense and far-reaching. The inference is as clear as day that an enormous influence must be exercised by procedures which impress such changes upon the caliber of the vessels, the action of the heart, and the composition of the blood in health; and that its effect in disease must be equally pronounced." The "musts" in this sentence are sufficient proof that the exact nature and influence of the changes are not yet sufficiently understood. Dr. Baruch has, we believe, at last impressed upon the profession, by his untiring persistence, the doctrine "that the antithermic uses of water are of far less value than its other qualities in febrile conditions," and that its real service is the stimulating influence upon the central nervous system, which is brought about by the reaction and manifested by the clearing up of those symptoms which we usually ascribe to febrile intoxication, but he also illustrates how firmly the antipyretic idea is impressed upon the medical mind by referring to "the good results in reduction of temperature" produced by ablution, the very first method described, and referring again to it in almost every other method.

In proof of his contention of the slightness of the antithermic result, he brings in evidence the slight reduction of the rectal temperature, which he regards as incomparably more accurate than the mouth or axillary temperature. This is a point upon which con-

siderably more investigation is required. His advocacy of the Brand method is undimmished in fervor, and the evidence he brings forward in favor of the reduction of mortality that follows its routine use is very convincing. As he justly remarks, the small experience of the average practitioner is practically worthless in comparison with the magnificent statistics obtained in various hospitals throughout the world. Even, perhaps, the inexact use of cold bathing in typhoid fever has produced a considerable decrease in the hospital mortality in this country, and for this also Baruch more than any other man is responsible, for he has taught the practitioners that cold bathing is good in typhoid fever, and they practise it without any very definite idea of the method. Dr. Baruch inveighs against the prejudice of the medical profession. We think it fairer to say the ignorance of the medical profession. By his own showing, there is only one medical college in the United States in which hydrotherapy is given adequate recognition. It cannot be supposed that the practising physician will look with favor upon a method of which he understands little or nothing; that is abused by quacks and charlatans, largely because of ignorance; and that requires either considerable personal effort on the part of the physician in educating members of the family, or the services of a trained nurse, who, perhaps, may need as much instruction. Much stress is laid upon the importance of accurate technique. In this we agree very heartily with Dr. Baruch. The indefinite directions given by the average practitioner are more calculated to do harm than good. The chapter on the hydriatic prescription deserves very careful and repeated reading.

In the special part the enthusiast is revealed. We are all, nowadays, ready to admit the paramount value of hydrotherapy in typhoid fever, but either the medical profession is difficult to convince or there is some error in the results obtained in cerebrospinal meningitis, pneumonia, Asiatic cholera, and in a variety of other diseases, even including chronic malaria. A reduction of 66 per cent. in the mortality of pneumonia, brought about almost exclusively by bathing, if correct, would make it imperative upon every practitioner to use this method. In functional nervous diseases and malnutrition its virtues are lauded.

The book concludes with a rather brief historical chapter. It can be commended as admirable in itself and almost the only adequate text-book on the subject in the English language.

J. S.

OPERATIVE MIDWIFERY. By J. M. MUNRO KERR, M.B., C.M. (Glasgow), Obstetric Physician to the Glasgow Maternity Hospital. Pp. 691; 294 illustrations. New York: William Wood & Co., 1908.

As stated by the author in his preface, it is many years since a book has been published in the English language dealing with operative obstetrics alone. In the period between the publication of Barnes' last edition in 1886 and the present, the whole obstetric viewpoint has been changed and the horizon enlarged, so that this department of medicine is now justly regarded as a surgical specialty. This may be looked upon as the author's apology for the publication of the volume, but after a most careful reading of the book we feel that none is needed.

The first twenty chapters, comprising 300 pages, are concerned with a consideration of dystocia, the remainder of the volume being given to a consideration of the operative technique. The first chapter of this latter portion gives a detailed account of the preparation of the patient, operating room, instruments, etc., while the next eight chapters treat of the various obstetric operations, such as the use of forceps, version, Cesarean section, and craniotomy. Then follow chapters on the manual removal of the placenta, abortion, placenta prævia, ectopic pregnancy, postpartum hemorrhage, and premature separation of the placenta. The last three chapters of the book are taken up with a study of the accidents to the mother and child. In an appendix of three pages an account is given of the method of determining the pelvic measurements by the Röntgen rays, which has been contributed by J. R. Riddell.

There are singularly few matters, either conclusions of the author or his method of presentation, with which the profession of America will not be in hearty accord. However, we cannot follow him in his suggestion as to the use of cocaine in certain types of cervical rigidity, nor in his advocacy of surgical anesthesia in primiparæ at the time of delivery of the head; in the former instance, because of the danger of infection and because there are better modern methods of meeting the indication; and in the latter, because of the danger of relaxation of the uterus, with resulting hemorrhage (the author calls attention in another portion of the work to the dangers resulting from a prolonged use of small amounts of anesthetic agents during the second stage). We also question whether American ophthalmologists will agree with his favorable prognosis in albuminuric retinitis, and we consider his method of closing the uterine wound after Cesarean section to be distinctly inferior. One of the most striking variations from the generally accepted teaching in this country is his statement that he does not consider the induction of labor of decided value in cases in which the true conjugate measures 8.7 cm. and above; he would limit its

employment to diameters between 8.1 and 8.7 cm. While we agree with him in the restriction applied to those below 8.1 cm., we feel that one of the most valuable indications for the operation is offered by diameters between 8.7 and 9.5 cm.

In considering the question of dilatation by the use of bags, we are glad to find that the use of the Pomeroy model is advocated, but we are sorry that the simpler model of Voorhees has not been at least mentioned as a substitute for the Champetier-de-Ribes. We must confess to an inability to understand the position assumed by the author when he advocates the vaginal Cesarean operation up to the twenty-fifth week, when rapid emptying of the uterus is demanded, but the abdominal method after this period. This is even more remarkable since the author believes in the operation in certain rare cases of eclampsia. We feel rather doubtful as to the correctness of the opinion that in face presentations it will be found that the posterior is the more usual position of the chin.

One of the most satisfactory chapters in the book is that upon the indications for, and the use of, the obstetric forceps. We are particularly pleased to note his universal use of the axis-traction principle, and his emphatic warning against the unjustifiable employment of forceps. He emphasizes the fact that brute force has no place in any obstetric operation, and strongly advises against the use of forceps in any case in which the conjugate measures less than 8.7 cm. He also demands that the head be well fixed at the brim, without overlapping, and that considerable time be given to allow of moulding. He is strongly of the opinion, which is fortunately becoming more general, at least among specialists, that cases with a more severe grade of deformity had best be delivered by some other method. His attitude here is the one consistently held throughout the book, as is shown repeatedly by his method of dealing with the various subjects considered, namely, that the midwifery of the future is to be a surgical specialty to be practised by men especially trained to deal with complicated cases. His chapter upon the metallic dilatation of the pregnant cervix is eminently satisfactory; indeed, we consider it as fair a presentation of the subject as could be written. He does not wildly enthuse over one special method, but, as the result of his own experience, he concludes that obstetrics has been advanced very decidedly both by the invention of Bossi and by the introduction of vaginal Cesarean section.

The presentation of the general subject of pelvic deformity is particularly to be praised, and his statement, when considering the indications for the special operations, that "if an operator says to me, before or early in labor, 'this is a case for symphyseotomy,' I invariably think he does not quite appreciate the refinements in the choice of operations for pelvic deformity," clearly reveals the attitude of judicial conservatism everywhere to be noted throughout the volume. As may be inferred from the above quotation, he is not

an advocate of symphyseotomy as a substitute for Cesarean section, forceps, or craniotomy, but believes that it still has a limited sphere, and also most emphatically believes that there are definite indications for the various methods of delivery, and demands, at least of the specialist, that these indications be respected. He is not impressed with the advantages claimed for pubiotomy, as contrasted with symphyseotomy, but does not consider that experience as yet justifies positive statements. His views as to the inadvisability of performing Cesarean section upon patients who have been subjected to unskilful attempts at delivery, or unclean examination before coming into his hands, are, as we understand them, those of the more rigid of the Continental and American schools, and seem to us possibly a little extreme, necessitating, as he admits, the not infrequent perforation of the living child.

In general, it may be stated that the work is a clearly expressed and scholarly production, well illustrated; that it comprises a most thorough and comprehensive consideration of all the matters germane to the work; and that a most delightful spirit of judicial fairness is in evidence throughout, without in the least leaving the reader in doubt as to the particular individual preferences and opinions of the author. The profuse references to the literature given in foot-notes throughout the book, together with the historical matter introduced, form a valuable and attractive feature of the work. W. R. N.

A Text-book of Minor Surgery. By Edward Milton Foote, M.D., Instructor in Surgery, College of Physicians and Surgeons, Columbia University, New York. Pp. 752; 407 illustrations. New York and London: D. Appleton & Co., 1908.

This volume upon minor surgery seems to fulfil, so far as the reviewer is aware, more nearly than does any other book of similar title the purpose for which it is intended. As a rule, authors have heretofore been satisfied to consider bandaging, ligations, amputations, excisions, and the like to be ample to write a book upon, even though the manuals and text-books include the same topics in their contents. Foote, however, has produced a book which discusses in detail the why and the wherefore of morbid conditions seen in the receiving ward and the surgical dispensaries, with their appropriate treatment. There are eight sections, comprising affections of the head, neck, trunk, genito-urinary organs, anus and rectum, arm and hand, leg and foot, and minor surgical technique. The injuries, inflammations, tumors, and deformities are separately considered in each section, and in the last section operative technique, bandaging, and the various surgical dressings are discussed. The principal

fault, it seems to the reviewer, is the failure of the author to appreciate the dividing line between the so-called minor and major parts of surgery. Perhaps this was inevitable if the book were to be of value, but it has led, through brevity, to many inaccuracies in the description of the pathology of some diseases. It hardly seems creditable that in modern times a surgeon should advocate non-interference with the lymph nodes in very early cases of carcinoma of the lip. No mention whatever is made of the value of the so-called Bier treatment for many of the conditions with which the author should be most familiar, nor is there any mention of the use of bacterins in the treatment of acne or persistent suppuration in certain localities. As an example of the sacrifice of exactness in order to conserve space, it may be mentioned that in discussing torticollis no mention is made of the spasmodic form which often requires nerve resections and is not amenable to tenotomy or manipulation, the only treatment referred to. Too much space is given, as is usual, to bandaging.

Notwithstanding these few criticisms, and perhaps many other imperfections which could be picked out here and there, the volume remains the best one upon its subject that has yet been written; and includes in its pages so many conditions which entirely escape mention in text-books upon surgery as to make it invaluable to the practising physician, the hospital interne, and the dispensary surgeon. Especial praise can be given the illustrations, which are not only new and clear, but are also accurately described by the legends. Attention should be drawn to the error on page 298, where a solution of nitrate of silver is recommended of the strength of 960 grains to the ounce.

G. P. M.

KIRKES' HANDBOOK OF PHYSIOLOGY. Revised and Rewritten by CHARLES WILSON GREENE, A.M., PH.D., Professor of Physiology and Pharmacology in the University of Missouri. Sixth American edition; pp. 723; 507 illustrations. New York: William Wood & Co., 1907.

A TEXT-BOOK OF PHYSIOLOGY. By ISAAC OTT, M.D., Professor of Physiology in the Medico-Chirurgical College, Philadelphia. Second edition; pp. 815; 393 illustrations. Philadelphia: F. A. Davis Co., 1907.

A TEXT-BOOK OF PHYSIOLOGY. By WILLIAM H. HOWELL, M.D., LL.D., Professor of Physiology in the Johns Hopkins University, Baltimore. Second edition; pp. 939; 281 illustrations. Philadelphia and London: W. B. Saunders Co., 1907.

THE aforementioned are new editions of well-known books on physiology. Each possesses features that have attracted to it a wide circle of readers. Kirkes' book furnished the pap that years ago

brought many mewling students of medicine on both sides of the Atlantic Ocean to professional maturity. Under the fostering hand of Dr. Greene, although the general arrangement of past editions has been adhered to, the quality of the present edition has been improved. The text has been largely rewritten, many new illustrations of physiological experiments and directions for laboratory work have been introduced, while the anatomical discussions have been curtailed. On the whole, the book may be cordially recommended—reflecting the best of the past, rejuvenated with new data of more recent times.

Dr. Ott's book has been much enlarged—by the addition of 240 pages and 250 illustrations. It has been much revised and considerably rewritten; the section on electrotherapy has been expanded, that on the sympathetic system entirely rewritten, and that on vision recast; less extensive alterations have been made elsewhere throughout. The book as a whole seems peculiarly adapted to the uses of Dr. Ott's own students.

Dr. Howell's book has undergone no fundamental change in arrangement or scope. Additions and changes have been made freely throughout the work, with the object of keeping the presentation of the subject abreast of the times, but as far as possible the additions have been counterbalanced by the elimination of such material as could be spared. If the book possesses features that distinguish it from others of its kind, these comprise evidences of breadth of view on the part of the author and his constant insistence upon the fundamental importance of basing the practice of medicine upon the teachings of physiology. The practical aspects of the subject are in evidence throughout the book—which can be cordially recommended as an interesting and philosophical treatise of value alike to the student and the practitioner. A. K.

PROGRESSIVE MEDICINE. A QUARTERLY DIGEST OF ADVANCES, DISCOVERIES, AND IMPROVEMENTS IN THE MEDICAL AND SURGICAL SCIENCES. Edited by HOBART AMORY HARE, M.D., Professor of Therapeutics in the Jefferson Medical College. Assisted by H. R. M. LANDIS, M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College, Philadelphia. Vol. IV, 1908; pp. 333. Philadelphia and New York: Lea and Febiger, 1908.

THE concluding volume for 1908 of *Progressive Medicine* opens with a statement of recent advances in diseases of the digestive tract by David L. Edsall. Among the important subjects discussed are a new swallowing sign of value in the diagnosis of stricture of the

œsophagus, the physiology of the digestive processes, methods of gastric analysis, the size and position of the stomach, acute dilatation of the stomach, gastric ulcer, gastric carcinoma, tests for occult blood, appendicitis, carcinoma of the appendix, intestinal tuberculosis, intestinal obstruction, pancreatitis, jaundice, cirrhosis of the liver, etc. J. Rose Bradford discusses diseases of the kidney, devoting special attention to tuberculosis, syphilis, chronic nephritis, bacilluria, pyelonephritis in pregnancy and the puerperium, etc. Joseph C. Bloodgood discusses shock, hemorrhage, anesthesia, wounds and their treatment, the surgery of the bloodvessels, of the muscles, and of the joints, and tumors. William T. Belfield treats of genito-urinary disorders—gonorrhœa, tuberculosis, tumors of the kidneys, renal calculi, diseases of the bladder and the prostate, etc. The volume closes with a practical therapeutic referendum by H. R. M. Landis, who clearly and succinctly sets forth the important advances in therapeutics during the preceding twelve months. One cannot say more of the volume as a whole than that it continues to justify its existence and to merit the confidence of the profession; it is a worthy successor of those that have gone before. A. K.

THERAPEUTICS: ITS PRINCIPLES AND PRACTICE. By HORATIO C. WOOD, M.D., LL.D., Emeritus Professor of Materia Medica and Therapeutics in the University of Pennsylvania. Thoroughly Revised and Rewritten by HORATIO C. WOOD, JR., M.D., Associate Professor of Pharmacology in the University of Pennsylvania, Philadelphia. Fourteenth edition; pp. 778. Philadelphia and London: J. B. Lippincott Co., 1908.

SINCE Wood's *Therapeutics* was a pioneer of its kind, and has been, for more than a generation, the standard by which similar books have been measured, criticism is disarmed by the passage of time and a splendid record. At present, therefore, it suffices to chronicle the publication of a new, the fourteenth, edition. Many minor and a considerable number of noteworthy changes have been made throughout the volume in an effort to make it fully representative of the present state of pharmacology and therapeutics; the chapter on cathartics has been completely rewritten, that on diuretics has been remodelled to make it correspond with present views on the physiology and pharmacology of the subject, and new chapters have been added on opsonic therapy and the ion theory. As an exposition of the objects and aims, the science and art of drug therapy, the book is unsurpassed and doubtless will long continue to enjoy, as it merits, the unbounded confidence of the profession.

A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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A Simple Method for the Serum Diagnosis of Syphilis.—TSCHERNOGUBOW (*Berl. klin. Woch.*, 1908, xlv, 2107) publishes a preliminary note on a simplified technique for the serum diagnosis of syphilis. The procedure is based on the following considerations: Fresh blood of a syphilitic patient contains the complement and the so-called syphilitic antibody in the serum. If extract of a syphilitic liver (syphilitic antigen) is added *in vitro* to such a blood diluted with a definite quantity of physiological salt solution, the mixture placed in a thermostat at 38° C. for one hour, and then inactivated serum, which is hemolytic for human red blood cells, is added, and the whole again placed in an incubator for two hours, there is formed a closed chain between the syphilitic antibody, the syphilitic antigen, and the complement, in which the complement is bound. Thus, the further addition of inactivated hemolytic serum is without effect upon the red blood corpuscles, since there is no available complement. If no syphilitic antibody is present in the blood, the complement remains free and hemolysis occurs after adding inactivated serum. The technique of the reaction is simple. One pricks the finger or ear of the patient, draws 0.1 c.c. of blood into a pipette, and dilutes it with about 1 c.c. of 0.9 per cent. sodium chloride solution. The Zeiss blood mixing pipette for white cells (1 to 10) is convenient for this purpose. The contents of the pipette are well mixed and emptied into a narrow test-tube (No. 1). Similarly, a second tube is filled (No. 2). Tube No. 3 contains blood diluted to the same degree with extract of a syphilitic liver in 0.9 per cent. sodium chloride solution. Tube No. 4 is prepared in the same way as No. 3. All four tubes are now placed in an incubator at 38° C. for one hour. At the end of this time 0.25 c.c. of inactivated serum hemolytic to human red blood cells is placed in Tubes 1 and 3. To

Tubes 2 and 4 a like quantity of 0.9 per cent. sodium chloride solution is added. All the tubes are again placed in an incubator for two hours and then in the ice chest over night. The result is noted the following morning. In Tube No. 1 hemolysis should always be found (if the serum contains complement). In the second tube there should be no hemolysis, as the red blood cells are in an isotonic salt solution. The result in the third tube will depend on the presence or absence of syphilitic antibody in the blood. If it is present the complement has been bound before the inactivated hemolytic serum was added, and no hemolysis occurs. If absent, the opposite result is obtained. The fourth tube simply serves as a control against any hemolytic action by the liver extract. Subsequently, Tschernogubow will describe the method in greater detail. It has proved trustworthy in his hands in a large number of cases. Its advantages are simplicity, the small amount of blood required, and the rapidity with which it can be carried out.

Precipitate Reaction with Lecithin, Sodium Glycocholate, and Sodium Taurocholate for the Diagnosis of Syphilis.—Wm. J. Butler and W. T. Mefford (*New York Medical Journal*, 1908, lxxviii, 822). Since the discovery of the serum reaction for syphilis many researches have been carried on to determine the identity of the substances contained in the extract used in the reaction, as well as to simplify the test. These researches have led to the use of other reactions in which, although the principle may vary, yet the result in each instance consists in a precipitate. Butler and Mefford have worked with the serum reaction and have compared the results obtained with it in the same cases, by the precipitate test. The performance of the latter test is comparatively easy and unattended with the many difficulties confronting one in making the complement-deviation test; it is made by mixing equal quantities of the serum to be examined and the precipitate reagent in a test tube and allowing it to stand at room temperature from fifteen to twenty hours. If the reaction is positive a precipitate will have formed and floats at the top of the fluid; on slightly tapping the tube it will descend in flocculi or whitish clouds. A turbidity of the fluid is not diagnostic. The results of these various tests in 74 cases examined may, according to these observations, be summed up as follows: The serum reaction surpasses in the percentage of positive results and in reliability the precipitate reactions for syphilis and parasyphilis. The glycocholate and taurocholate of sodium, especially the former, shows a large percentage of positive results in manifest syphilis, and a remarkably small percentage of positive reactions in control sera. The taurocholate gives the best results in positive reactions with parasyphilitic sera. The lecithin test is very unreliable and is definitely non-specific for syphilis. The results with active and inactivated sera in the control cases were practically alike with the glycocholate and taurocholate, but in the syphilitic and parasyphilitic cases an inactivated serum gave the greatest number of positive results, especially when the glycocholate was used. Butler and Mefford believe that more work will have to be done with these reactions before their real value can be determined, but in their examinations they fond the test satisfactory; few positive reactions were obtained with these salts in the controls examined.

Hemophilia.—Morowitz and Lossen (*Deut. Arch. f. klin. Med.*, 1908, lxxxxiv, 110) found in a case of hereditary hemophilia the usual prolongation of the coagulation time. From their studies upon a patient's blood they have shown that the delay in the coagulation time is to be attributed to insufficient formation of the fibrin ferment factors, especially the thrombokinase which is derived from blood platelets and leukocytes. That this is the only etiological feature of importance they do not assume. By means of suction applied to the skin of the back they were unable to demonstrate any greater liability to hemorrhage in their patient than in three normal adults examined.

Diphtheria Bacillus Septicemia.—Ucke (*Fortschr. der Med.*, 1908, xxvi, 1005; *Zentralbl. f. Bakt.*, Band lvi) describes a patient with impure heart sounds, frequent pulse, trace of albumin in urine, an area of inflammation without fluctuation in the right gluteal region. The patient had had high fever for two weeks preceding admission to the hospital. An angina with membrane had preceded the inflammation of the gluteal region, and Loeffler's bacillus was found in the throat. Operation on the right gluteal region was without result at first and blood culture was negative. No result followed the injection of 300 units of anti-diphtheritic serum. At a subsequent dressing of the wound, however, large quantities of pus discharged through a fistulous opening. In the pus, diphtheria bacilli were found; their virulence was greatly diminished, as animal experiments showed. A second blood culture gave a pure growth of diphtheria bacilli. Only three similar cases are recorded in the literature.

Chyluria.—Magnus-Levy (*Ztschr. f. klin. Med.*, 1908, lxvi, 482) reports his study of a case of chyluria. The patient was a man aged fifty-one years, from Western Prussia. Six years previously he had noted that his urine was white and turbid during the winter, but in the spring and summer, became clear. The condition had recurred regularly each winter since then. The only complaints were pain in the back, especially on the right side, and pain on micturition. At times clots were voided. In January, 1908, the patient complained of thirst, loss of weight, and polyuria. Urinary examination revealed chyluria and diabetes mellitus. The night urine was chylous; the day urine clear. On reclining during the day and sleeping upright in a chair, the urinary finding was reversed. The withdrawal of fat from the diet led to clear urine at night, but the other constituents of chyle, such as albumin and lymphocytes, were still present in the night urine. With increasing quantities of fat in the food, a corresponding increase in milkiness of the urine occurred. Fibrinogen, albumin, and globulin were determined by fractional precipitation with ammonium sulphate. Cystoscopy revealed the fact that the chylous urine came entirely from the right ureter. The urine was collected from the two kidneys by catheterization of the ureters and analyzed separately. By computation Magnus-Levy shows that the proportion of fat, albumin, and sodium chloride in the chylous urine corresponded exactly with the composition of chyle as given by Munk and Rosenstein. No ova or embryos were found in the urine, nor was the blood serum fatty. There are two theories as to the origin of chyluria: (1) That the condition

results from direct addition of chyle to the urine, and (2) that the abnormal constituents are derived from the blood, no communication existing between the urinary and lymph passages. A defective catabolism of the chylous material is assumed. If the latter theory were correct, all cases of chyluria should be bilateral. Furthermore, direct communication has been found in a certain number of cases. In two-thirds of all the cases chyluria disappears either in the reclining or upright posture, and that would be impossible if the blood condition were the cause of the chyluria. The constant finding of lymphocytes in cases in which centrifugalized urine is examined also points to direct admixture of chyle. If one extracts sufficient urine with ether, cholesterin and lecithin are found. Their quantity in the chyle depends largely upon the diet. Again, the absence of glycosuria in chyluria has been urged as an argument against direct admixture of chyle to urine. But, as the author points out, Munk and Rosenstein have shown that chyle contains about 0.1 per cent. sugar on a fat and proteid diet; 0.3 to 0.4 per cent. after a carbohydrate meal. Only in the latter condition would one expect to find a glycosuria. The absence of clotting in some cases is also without significance, for in certain instances lymph and chyle fail to clot. Magnus-Levy assumes that in all cases a direct communication must exist between the lymph vessels and some part of the genito-urinary tract. The absence of chyluria for months and years at a time might be explained by the closure of the opening, the widening of the lymph vessels, or the establishment of collateral channels. The daily intermissions must be explained on purely mechanical grounds. This explanation of chyluria holds good both for parasitic and non-parasitic cases.

A New Test for Bile Acids and the Detection of Bile Acids in the Urine.—Since Pettenkofer's test is not specific for bile acids, giving a red color with albumin, urea, carbohydrates, fatty acids, etc., it is necessary in applying it first to separate the bile acids in the urine. Confusion may also arise in the spectroscopic examination. Therefore, a new test for the detection of these acids is desirable. JOLLES (*Hoppe-Seyler's Zeit. f. physiol. Chemie*, 1908, lvii, 30) has devised a test for bile acids in which the reagents used are 5 per cent. rhamnose solution and concentrated hydrochloric acid. If one adds one to two drops of rhamose solution to 2 to 3 c.c. of dilute (0.1 per cent.) solution of taurocholate or glycocholate of sodium and then an equal amount of concentrated hydrochloric acid to the mixture a rose color appears on gently boiling. This color soon disappears, and after standing a short time a beautiful green fluorescence supervenes. If the same experiment be repeated with 1 per cent. solutions of taurocholate and glycocholate, boiling produces a deep red color. On continued boiling the fluid appears reddish brown with transmitted light, malachite green with direct light. Neither glycochole nor taurin gives this reaction. It is, however, given by cholalic acid as shown by the following experiments: When two drops of 5 per cent. rhamnose solution and 2 c.c. of concentrated hydrochloric acid are added to 2 c.c. of 0.1 per cent. alcoholic solution of cholalic acid (Merck), a white cloud appears from precipitation of the acid. On warming the mixture a red color develops, changing into a beautiful green fluorescence on boiling. A substitution of sulphuric acid for

hydrochloric gives less satisfactory results. From the rhamnose solution Jolles has found that methyl furfurol is formed by the action of the hydrochloric acid. A solution of methyl furfurol may be substituted for the rhamnose solution without altering the results of the test. But there is no advantage in doing this. In solutions the brilliancy of the fluorescence is dependent upon the concentration of the bile acids and, therefore, with very small quantities of cholalic acid the fluorescence is diminished. The minimum amount of cholalic acid in 1 c.c. of alcohol which can be detected by adding 1 drop of 0.1 per cent. rhamnose solution and 0.5 c.c. of concentrated hydrochloric acid varies between 0.005 and 0.001 gram. To render the green fluorescence more marked 1 to 2 c.c. of ether may be added after cooling and the contents of the test tube shaken. The fluorescence is then seen in a watery solution. None of the confusing substances with Pettenkofer's test will give a positive reaction with Jolles' test. For the recognition of bile acids in the urine one adds 15 c.c. of 3 per cent. casein solution (3 grams casein in 100 c.c. of water) to 50 c.c. of urine. This is well mixed and 10 per cent. sulphuric acid is added drop by drop (usually 0.6 to 0.8 c.c. is sufficient) until all the casein is precipitated. An excess of sulphuric acid is to be avoided. The contents of the test tube are now filtered and the precipitate is washed into a beaker with 100 c.c. of absolute alcohol and allowed to digest at ordinary temperature for about one hour. This is then filtered and 4 to 5 c.c. of the filtrate are placed in a test tube with one drop of 5 per cent. rhamnose solution and 4 to 5 c.c. of a concentrated hydrochloric acid. The mixture is heated to boiling and the boiling continued for one to two minutes. After cooling the contents of the test tube about 2 c.c. of ether are added and the contents shaken. In the presence of bile acids the characteristic green fluorescence is seen. The test will detect as little as 0.5 per cent. of sodium taurocholate; in concentrated urines and those rich in indican and aromatic oxy acids the test is less delicate.

The Presence of Mydriatics in the Urine.—DIEM (*Deut. Arch. f. klin. Med.*, 1908, lxxxiv, 174) has repeated the work of Pal, testing the urine of nephritics and others for mydriatics on the enucleated eye of a frog. The work of Schur and Weisel seems to point to an increased quantity of adrenalin in the blood of nephritics. Diem has tested the urine in a great many patients and finds that of a little over one-half the nephritics examined produced a marked widening of the pupil in the frog's eye. This phenomenon is not, however, characteristic of nephritis, being observed with almost equal frequency in other diseases. The substance which caused the mydriasis could not be determined, but Diem thinks various factors are concerned; that it is possible that substances are present in many urines which inhibit mydriasis; and that it is highly improbable that the test is specific for adrenalin in the urine.

Specific Stimulation of the Intestinal Peristalsis by Intravenous Injection of "Peristaltic Hormone."—ZUELZER, DOHRN, and MARXER (*Berl. klin. Woch.*, 1908, xlv, 2065) were led to the study of intestinal peristalsis by the work of Starling on the mammary gland and that of Bayliss and Starling on secretion. They suspected that a hormone existed in the

stomach which acted as a specific stimulus to intestinal peristalsis, and they succeeded in demonstrating such a body in the gastric mucosa, to which they gave the name "peristaltic hormone." To obtain it the animal must be taken at the height of digestion. It is not obtained from the fasting stomach. This hormone, like others, acts through the blood. If one injects it intravenously in the rabbit, an energetic contraction extending from the duodenum to the rectum begins in a few seconds. To prepare the hormone, they extracted the gastric mucosa with salt solution or dilute hydrochloric acid and precipitated the albumin with alcohol. The stomachs of various animals, the rabbit, pig, horse, and cattle, contained the hormone. In cattle it is only found in the glandular part of the stomach. A similar hormone, weaker and not constantly present, may be obtained from the duodenal mucosa. In a subsequent communication the authors will consider the therapeutic uses of this body. [The possible importance of this discovery from a therapeutic standpoint is easily appreciable.—W. S. T.]

The Ethereal Sulphates in the Urine in Auto-intoxications.—R. BRUNON and M. GUERBET (*Presse médicale*, 1908, lxxxv, 673) have noted previously that the proportion between the ethereal sulphates and the total nitrogen may be of great service in the diagnosis, treatment, and prognosis in cases of auto-intoxication. In the normal individual the quotient obtained by dividing the amount of ethereal sulphates by the total nitrogen gives figures usually varying between 1 and 1.4; whereas, in the auto-intoxications this is greatly increased. Brunon and Guerbet report in detail their observations on 15 cases of auto-intoxication, followed closely as regards this relation, and in all they found the quotient higher than normal, and up to 3.16 (some authors report it as high as 6 to 7), but as the signs of the auto-intoxication diminish, the quotient became smaller, until, when cured, the patients showed a normal quotient again.

A New Microchemical Test for Mucus in the Feces.—HECHT (*Wien. klin. Woch.*, 1908, xxi, 1554) employs a 2 per cent. solution of brilliant green and a 1 per cent. solution of neutral red. These are mixed in equal parts. The resultant solution is about the color of Ehrlich's triacid stain. A small drop of the mixed stain is added to a bit of fecal material on a slide, mixed well, and covered with a cover-glass. The preparation is pressed with filter paper to remove the excess of stain, and is then ready for examination. The stool is stained diffusely green, the fluid being red from withdrawal of the green. The mucus is precipitated and stained a brilliant red; fibrin is stained bluish green, likewise the protoplasm of cells, and especially the cellular elements, which have imbibed alkaline soaps. In addition to mucus, bacteria, cell membranes of vegetable cells, and nuclei of cells are stained red. Clumps of bacteria can, however, easily be distinguished from mucus by using high magnification. Particles of mucus that are difficult to demonstrate otherwise are easily recognized by this method.

SURGERY.

UNDER THE CHARGE OF

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Drainage of the Brain Ventricles by Transplanted Bloodvessels.—PAYR (*Archiv f. klin. Chir.*, 1908, lxxxvii, 801) reports three cases operated on for internal hydrocephalus, by making a drainage path from the ventricle to the venous current by means of a transplanted bloodvessel. The essentials of the operation consist of a water-tight conduction of the cerebrospinal fluid toward the venous path, the preservation of the normal venous circulation, the prevention of a back current of blood into the ventricle, the avoidance of hemorrhage and air emboli during or after the operation, and the avoidance of infection. When a vein, as the long saphenous, is employed for the anastomotic tube, the function of the valves must be taken into account. The longitudinal sinus should be employed as the venous trunk into which the anastomosis is to be made. Failure of the operation may result from insufficient length of the transplanted segment of bloodvessel or from unsuitable quality of the vessel (phlebosclerosis, varicosities). In some cases of congenital hydrocephalus the longitudinal sinus may be absent, or it may be obliterated by inflammation or thrombosis. Severe hemorrhage may compel one to abandon the operation, or it may fail from a poor line of sutures, primary or secondary compression, or a false direction of the transplanted bloodvessel. Necrosis of this vessel may result from disturbed nutrition, or it may follow mechanical, chemical, or thermal insult. Thrombosis of the longitudinal sinus or the drainage path may occur. The transplanted vessel may be forced out of the ventricle, or softening of the brain in the region of the drainage vessel, or secondary infection, may occur. A permanent drainage of the ventricle toward the venous current is a method of treatment not only for the internal hydrocephalus, but also for the pressure on the brain. This is of especial importance for those cases in which the cause of the condition cannot be recognized or cannot be overcome. This drainage is rendered possible, probably, because the transplanted bloodvessel is an endothelial lined passage between the ventricle and the sinus. The chief dangers, infection, air emboli, and hemorrhage at the time of the operation or later, are best avoided by asepsis, measures for provisional hemostasis, and exact suturing. There must be some provision for a valve to prevent a back flow of blood into the ventricle. When the cerebrospinal fluid cannot be drained into the blood path, one must be contented with drainage into the subarachnoid space or into the soft tissues of the scalp.

The Treatment of Operative Pneumothorax in a Healthy Pleural Cavity.—LINE (*Archiv. gén. d. chir.*, 1908, ii, 335) says that a total pneumothorax from an external cause and due to a small opening in the pleura is nearly always benign, provided that the wound has not been infected. When there is a large tear of the pleura, the respiratory difficulties which follow the entrance of air vary in intensity according to the conditions of the experiment. They are not relieved by the diminution of the bleeding area, but depend upon the degree of dislocation of the mediastinum. Total pneumothorax is always borne well if it is produced slowly and carefully, and if the opposite lung functionates well. In the course of operations by the transpleural route, the employment of apparatus for negative or positive pressure seems to be difficult to carry out, while the true surgical measures are always sufficient to provide against the respiratory difficuties which follow the entrance of air into the pleural cavity. The patients do not die during the operation from pneumothorax, but from the infection of the pleura; so that it is not so much the measures to prevent pneumothorax that one should seek, as those of avoiding infection. If the pleura has become infected, one should provide against its consequence, as much as possible. The cavity should be tamponed with warm compresses and the field of operation isolated, as in operations in the abdomen. If the symptoms become alarming, the packing of the pleural cavity should not be insisted upon, nor should too much be expected of artificial respiration. It is important that the resorption of the air be accomplished quickly, to empty the cavity and eliminate the dead spaces in which the liquids can stagnate and become infected. If, in spite of the precautions taken to avoid infection, the early respiratory phenomena persist during the following days coincidently with fever, one should lose no time in draining the pleural cavity by a costal resection. It would, perhaps, be an advantage to provide irrigations also. With a good technique and rigorous asepsis, provoking the pneumothorax slowly and scientifically, watching the patient carefully, and being ready to drain on the least rise of temperature, one can always carry out an operation by the transpleural route, without having recourse to resources other than those which surgery offers, and without recourse to negative or positive atmospheric pressure.

Concerning the Anatomical Condition of Suprarenal Tissue Transplanted into the Kidney.—STOERK and HABERER (*Archiv f. klin. Chir.*, 1908, lxxxvii, 893) have further investigated the fate of suprarenal tissue embedded in the kidney, Haberer presented a paper on this subject last year, the object of which was to obtain experimental evidence concerning the question of the origin of renal tumors, which, according to the hypothesis of Grawitz, take their origin in fœtal life in a portion of the suprarenal gland which becomes developed in the renal cortex; also concerning the origin of the so-called hypernephroma. In the sense of providing a suitable nourishment, a suitable method of transplantation will protect against destruction of a portion of the suprarenal gland, as well the cortical as the medullary portion. From this embedded portion there is developed, by the combination of the newformed and old altered tissue with the continued good nourishment, a new organ, which agrees in function and structure with the physiologi-

cal suprarenal gland. In other respects it deviated in a characteristic manner from the original gland. The proliferation of the new and old cortical cells produces a histological picture which completely agrees with the so-called adenoma of the suprarenal cortex of man and animals. In no aspect do these formations agree with the histological picture of the renal tumors of Grawitz.

A Typical Fracture of the Internal Epicondyle of the Femur.—VOGEL (*Archiv f. klin. Chir.*, 1908, 1076) says that Stieda, two years ago, called attention to a new fracture, which consisted of a thin, flat piece of bone, separated from the internal condyle. It is shown by the skiagraph to remain more or less closely applied to the condyle. Its position corresponds to the lowest part of the insertion of the adductor magnus and the origin of the internal head of the gastrocnemius. Stieda thought that it was due either to a tearing off by the muscle or to direct violence. In Stieda's cases the history seemed to show that it was caused by direct violence. Vogel's two cases likewise showed the same cause, and he thinks that it is not due to muscular pull. The symptoms consist of the typical pain at the seat of fracture, swelling, ecchymosis, and limitation of movement in the joint. The most characteristic symptom in recent cases is the tenderness which is always precisely located. The skiagraph will confirm the diagnosis. The treatment up to the present time has always been non-operative. Vogel has obtained tolerably good results from the use of Bier's hot-air treatment, combined with massage and passive movements. In one case he cut down upon and removed the loose fragment. The operation is without danger, can be done in five minutes and does not open the joint, at least in the typical fracture. In two cases, following the conservative treatment, the patient experienced some disturbances afterward, and the skiagraph showed small osteophytes or exostoses, which resulted from the stripped up periosteum. These may disappear later, but they may also increase in size, so that their sharp points may give rise to greater disturbance. The patient from whom the fragment was removed by operation at the time of discharge did not complain either of pain or any objective abnormality. The prognosis of the injury is, in general, good, but it should take into consideration the possibilities of the late results of conservative treatment. It is of course possible for consolidation to fail and the fragment to become absorbed. It may require a long time before the patient is able to work. With the removal of the fragment, the treatment will surely be more brief and the guarantee of complete recovery may be more complete than with the conservative method.

Osteoplastic Compensation after Resection of the Lower Jaw.—RYDYGIER (*Zentralbl. f. Chir.*, 1908, xxxv, 1321) does not believe that the filling in of the defect in the lower jaw left by resection can be done as well by a piece of rib carried upward in a skin and fascial flap as by a similar flap carrying a piece of the nearer lying clavicle. He has been doing the latter operation for sixteen years. A skin flap is formed over the clavicle, reaching two fingers' breadth below its lower border and wide enough to suit the deficiency in the jaw. The flap is freed only up to the lower edge of the clavicle, but it is not separated from the anterior surface of the bone. Considerable care will be necessary to avoid

this separation, as the subcutaneous tissue is very loose. The anterior half of the clavicle is then separated from the rest of the bone and lifted with the flap, which has an attachment at its base above. The lower loose portion is then wrapped around the under surface of the separated piece of bone, and eight days are allowed to pass for the development of firm adhesions between the bone piece and its enveloping flap. It can then, by means of a sufficiently long skin flap above, be transplanted into the defect in the lower jaw.

The Treatment of Biliary Lithiasis.—BENOIT (*Jour. d. méd. et d. chir.*, 1908, iii, 329) gives a *resume* of the teachings of Gilbert, Carnot, and Jomier on the medical treatment, as well as that of Mongour on the surgical treatment, of gallstones, these men representing the French school. The arthritic diathesis predisposes to gallstones. The local conditions favoring their formation are infection of the gall-bladder and relative stagnation of the bile. In women, in addition to the usual causes of bile stagnation, there is the use of corsets. Preventive medicine depends more upon hygiene than upon drugs. There is no doubt that the alkaline salts, bicarbonate of sodium in particular, will aid in keeping the bile in a fluid form. Calomel, by its cholagogue action, tends to combat the torpor of the liver, but cannot be kept up long because of its disturbing effect on the digestive tract. Hygiene is most important, and the diet should be especially lactovegetarian. Hydrotherapy in all its forms favors elimination by the skin, as well as frictions, massage, and sports not to excess. In the greater number of the cases one is justified in provoking or aiding the migration of the calculus, as by calomel and sodium sulphate, which increase the biliary flow, and by salicylate or benzoate of sodium, which are cholagogues and antiseptics. Olive oil is advised for stones in the common duct. The remarkable toleration of the biliary tract, and the good effect of therapeutic measures, will often render surgical action unnecessary. Mongour, reporting on the surgical side, said that operation should be a therapeutic exception. Those who argue for early operation, that is, on the first attack of colic, are not justified by the facts or results. Biliary lithiasis is not, exclusively, a local affection, and operation does not affect the diathesis. In spite of possible complications, lithiasis ought to be considered a benign affection, as shown by statistics from Vichy and those published by the International Congress of Insurance Companies. The operative death rate, even in the more favorable cases, is more than 2 per cent. It is greater than that if one takes into account the late postoperative accidents. Finally, operation does not insure the patient against recurrences. When the stones are in the gall-bladder, the indications, as formulated by Mongour, are as follows: Exceptionally frequent and painful cases; when the patient cannot follow a prolonged medical treatment; in infection of the gall-bladder, especially in chronic cases; in cases of general infection starting from the gall-bladder. If the stones are in the common duct, the time of operation depends upon: (1) the quality of the biliary secretion, and (2) the nature and intensity of the local or general infectious complications. Operation should be done when the bile appears in the urine, and can be deferred as long as there is only a simple jaundice.

The Prevention of Kidney Stone Recurrence.—KLEMPERER (*Deut. Zeitschr. f. Chir.*, 1908, xcv, 304) says that the recurrence of renal calculi after they have been removed by operation is well known. An abundant free flushing of the kidney and its pelvis will prevent the deposit of calculous particles. The original nucleus is of microscopic size, and its increase in volume depends upon the apposition of the urinary sediment, which is possible only in a slowly escaping or stagnating stream. The most important point in the treatment, therefore, is to administer large quantities of liquid by the mouth. The drinking of 200 or 300 c.c. of fluid, every two or three hours, and especially a large quantity before going to bed is advised. The night urine is particularly inspissated and prone to sedimentation. Still more fluid should be drunk during the night, especially by old people, who frequently get out of bed. The kind of drink is not very important, the alkaline, carbonic acid, mineral waters being preferred. Even alcoholic drinks may be taken. Obesity and heart weakness favor calculi formation. Von Örtel and Schweninger believe that much drink increases the obesity. Experience and physiological experiments, however, have shown that much fluid in an empty stomach favors emaciation, so that fat people should not drink during meals. Patients suffering from cardiac disturbances should take fluid during periods of disturbed compensation more cautiously. According to Klemperer's observations, the stones in these cases are small and round, and are passed without much difficulty. A mixed diet is preferable to a uniform one, which favors the formation of an acid urine and, therefore, an increased sediment. An alkaline reaction due to the administration of the carbonates can favor the deposit of phosphates. In his collection of kidney stones are several in which the acid nucleus is coated with a white, phosphatic layer. Patients with urate or oxalate calculi should take a moderate amount of meat with vegetables and fruits. Medicines are usually of little value. In cases with oxaluria, however, small doses of magnesium sulphate, which are excreted by the urine and can take up calcium oxalate in solution, should be given. When the alkalinity of the urine is due to infection, of more importance than diet or urinary antiseptics is the mechanical effect of flushing the kidneys or the removal of any existing obstruction.

Diffuse Septic Peritonitis Due to Appendicitis.—FOWLER (*Annals of Surgery*, 1908, xlviii, 828) says that we must look for a lowering of the high mortality rate in early operation rather than in any further development in mechanical intervention. Early postural drainage is of greater aid in preventing septic material from reaching the diaphragmatic peritoneum than in preventing further absorption after this area is once involved. The manner of instituting postural drainage matters little, provided the pelvis is sufficiently low for gravitation to take place and the patient is comfortable. Peritoneal lavage dilutes septic material, and when practised should be continued until the cavity is partially closed. Plastic lymph not removed by irrigation or by simple lifting should not be disturbed. All cases of this nature should be drained. The ideal method, in women, is by a posterior colpotomy incision, by means of a large rubber tube. Cases not drained frequently develop pus pockets and superficial wound infections. Ochsner's

treatment should be instituted after operation and Murphey's proctolysis practised. Open the abdomen by a small incision over McBurney's point, deal quickly with the primary focus, prevent evisceration, and use the greatest gentleness in handling parts.

Simultaneous Ligation of Both External Iliac Arteries for Secondary Hemorrhage Following Bilateral Ureterolithotomy.—Moschowitz (*Annals of Surgery*, 1908, xlviii, 872) reports a case in which a left-sided ureterolithotomy had been done by another surgeon June 15, 1907. Eleven days after this operation an attack of right-sided renal colic occurred, and was regarded by the surgeon in charge as an example of contralateral pain. On July 20, 1908, the patient was operated on a second time by Moschowitz. This operation consisted of a bilateral ureterolithotomy, with the removal of two calculi from the pelvic portion of each ureter, through lateral extraperitoneal incisions. The ureteral incisions were closed by interrupted iodine catgut sutures. Seven days after operation, on removal of the left rubber drainage tube, which came away easily, a tremendous hemorrhage followed immediately. A finger introduced promptly controlled the bleeding at once. Immediate operation showed a hole in the external iliac artery the size of the tip of the little finger, produced by the pressure of the tube. Accidental pulling out of the tube on the other side, after the ligation of the left artery, resulted in a similar hemorrhage on that side. The same cause was found and the same measures for its control were resorted to. All pulsation ceased below the ligature and both lower extremities became blanched. These were wrapped in cotton and the trunk and legs were slightly elevated. Yet the general condition was fair, and on the evening of the same day the toes were warm, of a delicate pink color, and capable of slight active motion. On the following day slight femoral pulsation was to be felt, and on the third day an occasional flutter in the dorsalis pedis artery. Thereafter recovery was entirely uneventful. Ten weeks after operation, the incisions were firmly healed; there was no hernia; pulsation could be felt in both femorals and dorsalis pedis arteries, although somewhat smaller than in the normal. This is the first case reported of simultaneous ligature of both external iliacs, successful or otherwise.

THERAPEUTICS.

UNDER THE CHARGE OF

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Principles of the Dietetic Treatment of Severe Diabetes.—Kolisch (*Zeit. f. Phys. und Diat.-Therapie*, 1908, iii, 150; iv, 212) discusses the etiology and pathology of diabetes and considers the dietetic treat-

ment of severe cases. He considers it most important to reduce the diet so that it will supply the smallest possible number of calories necessary for the maintenance of the body weight. He believes that the number of calories needed for a diabetic patient is less than for a normal person, if the diet is properly chosen. Thus, he has found that a diabetic will not lose weight, but frequently increase in weight and strength, on a diet which is chiefly vegetarian. The number of calories per kilogram of weight which he considers necessary is about 20. A second principle is that the proteids should be reduced in amount. He thinks that proteids when taken in large quantities are capable of breaking down the body cells and of causing an increased glycosuria. The third principle is that the diet should contain the maximum amount of carbohydrates that can be used without increasing the glycosuria. This is effected by a reduction of the proteids, since it is known that a diminution of the proteid in the diet makes possible an increase of the carbohydrates, and vice versa.

Experimental proof of this teaching was given in the report of a series of severe cases by Kolisch and Sheeman-Lequerc. Each of the patients was put on a moderate standard diet until the output of nitrogen and the glycosuria became constant. Then the amount of carbohydrates was increased and the proteids diminished, the value in calories remaining equal, with the result that the glycosuria did not increase and in some cases diminished considerably. When the carbohydrates were given in large excess of calories, as compared with the amount of proteid withdrawn, the glycosuria increased considerably, but on a return to the standard diet the level of the glycosuria fell below the level of the first standard period. The principles advanced by Kolisch will explain the results obtained by the milk, buttermilk, rice, oatmeal, and potato treatment of diabetes. These diets have the common characteristics of being of relatively low caloric value, of containing little proteid, and, with exception of the milk, the proteid is of vegetable origin. The carbohydrate content of these diets is relatively high, and, moreover, the proteids and carbohydrates are given constantly in the same form which makes them better tolerated by diabetic patients. Kolisch has used a vegetable diet for ten years. For the average case, about 1500 calories per day are necessary. He claims the main advantages are as follows: (1) The largest possible amount of carbohydrates can be taken because of the low proteid content. (2) The proteid is vegetable proteid, and therefore less harmful. (3) Vegetables contain relatively a large amount of alkalies which are of service in preventing or in treating acidosis when it exists. (4) A vegetable diet helps peristalsis. Small quantities of egg albumin and fat are added to the vegetable diet. He gives the following standard diet, to which gradually increasing amounts of carbohydrates may be added as the glycosuria diminishes: For breakfast: Cup of coffee with 60 gms. whipped cream, white of one hard-boiled egg. At noon: (Karfiol) Milk, butter, cabbage with bacon, white of one egg, salad (endive, lettuce, cabbage) with 20 gms. oil, one apple. For supper: Tea or coffee with 60 gms. whipped cream, mushrooms with fat, white of one egg, salad, or beans with butter, one apple.

The Influence of Prolonged Digitalis Treatment on the Normal and Diseased Heart.—CLOETTA (*Therapie der Gegenwart*, 1908, x, 437) relates

the results of some very interesting experiments with the prolonged use of digitalis on animals. He treated healthy dogs continuously for two years with gradually increasing doses of digitalis given subcutaneously. None of these dogs showed any cardiac hypertrophy or other anatomical or functional changes in the circulatory organs. His results showed that a functionally weak heart was not made strong, except during the period of taking the drug. The most interesting results were obtained in the animals in which he induced an acute endocarditis of the aortic valves and produced an aortic insufficiency. Some of these animals were then treated with digitalis, and after several months their hearts were compared with the control animals. These experiments seemed to indicate a decidedly beneficial influence from the use of digitalis upon the late results of an acute endocarditis. This influence was shown not only by the anatomical differences, but also by the functional capacity during life in the two sets of animals. The hearts of the digitalis animals were only 30 per cent. larger than normal, while in the untreated animals the hypertrophy was 80 per cent. above the normal. The fact that the lesion induced was an aortic insufficiency makes these results particularly interesting. Cloetta believes that the use of digitalis kept the hypertrophy within bounds and maintained the circulation in a more normal manner in the digitalis animals. It is needless to say there was no effect upon the valvular defect. Cloetta believes that in acute endocarditis the heart is apt to be irritable and does too much or too little work, that the good effects of the digitalis are due to its soothing and quieting action, and that there will result less dilatation and, in consequence, less subsequent hypertrophy. Several of the untreated animals died with dropsy in less than six months, while none of the treated animals showed any signs of heart trouble. From these results Cloetta is convinced that digitalis is bound to have a favorable effect upon recent endocarditis. He advises that since digitalis managed carefully is harmless, it should certainly be tried, and he believes that if this is done early, there will be less need of the drug in later years.

The Treatment of Arteriosclerosis.—SHEFFER (*Arch. gén. de méd.*, vi, 337) affirms that the generally accepted belief that arterial hypertension is the cause of the vascular lesions of arteriosclerosis cannot be considered absolute. He holds this belief because a marked arteriosclerosis may exist without hypertension. Danin found hypertension in 70 per cent. of his cases, Grödel in 65 per cent., and Strasburger in 46 per cent. Sheffer himself found hypertension in 60 per cent. Lancereaux, Hayem, and Chantemesse consider that hypertension is the result, and not the cause of arteriosclerosis. In either case the arteriosclerosis is due to the same causes, the injurious effect of poisons or toxins in the circulating blood. These poisons produce either vasoconstrictions or degenerative changes directly in the walls of the arteries. A necessary factor is an hereditary or individual predisposition. Sheffer believes that this predisposition consists of a defect in the metabolism of silicates. Gaube, Robin, and Binet have shown that the silicates are important in the economy. The quantity of silica in the muscles of an average man is about 6 gm., or twice as much as the amount of iron in the blood. Decene believes that the silicates prevent the deposition of lime salts. In the treatment of arteriosclerosis Sheffer advises

the use of sodium silicate, which is a thick, syrupy liquid, with a disagreeable taste, and therefore should be given well diluted. The average daily dose is 1.5 to 3 grams. It may be employed for any length of time, although it is better to suspend its use from time to time. No digestive disturbances have been observed from its use; in fact, the digestive function seems to be improved. The arterial tension is lowered and remains at a constant and lower level after the drug has been used for from two to four weeks. The symptoms of cerebral arteriosclerosis, headache, and dizziness disappear very rapidly, and albuminuria diminishes or may entirely disappear. This result is also obtained in cases in which there is no increase of tension. A longer time is required to relieve dyspnœa or angina pectoris. In every case the general symptoms are improved and the patients feel stronger. The best results are obtained in the presclerotic stage. Sheffer also claims good results with this medication in the treatment of arthritis deformans.

The Treatment of Syphilis.—WECHSELMANN (*Fol. Therap.*, 1908, iv, 122) reviews the treatment of syphilis which has been placed on a more scientific basis by the discovery of Spirochæta pallida and by experimental investigation. Various investigations seem to prove that mercury acts directly upon the syphilitic virus. Neisser was able to diminish the eruption of the disease in apes inoculated with syphilis if mercury was introduced into the animal simultaneously with or soon after the inoculation. Atoxyl seems to act in the same way, but the doses required are frequently too large to be entirely safe. Moreover, atoxyl is not nearly so efficacious as mercury, and the effect of atoxyl on the late stages of syphilis is still unknown. Iodine appears to act on the tissues as an absorbing agent and only slightly on the syphilitic virus. Since spirochetes have been found in gummas and the infectious nature of these gummas has been established, the use of mercury in the tertiary stage of syphilis is justified. Wechselmann believes that the internal administration of soluble salts of mercury is weak in action, while the injection of insoluble salts has a much more energetic healing effect. However, the injection treatment frequently causes severe toxic symptoms and sometimes fatal results. He believes that the inunction method is just as potent and less dangerous. When the inunction method is inapplicable, the injection treatment is indicated. For this purpose mercury salicylate and thymolacet are the most suitable salts. The average dose is 0.1 gram every eighth day. Gray oil acts more energetically, but is more painful and has a greater tendency to toxicity. It should be used only in the rarer cases which resist ordinary treatment. Sterilized paraffin oil, olive oil, or vasenol may be employed as vehicles to convey the mercury. The injections should be made into the upper and outer quadrant of the buttock, where the veins are few in number. The injection of soluble salts is only suitable for the milder cases or for intermittent treatment. They are apt to be more painful. He recommends for internal medication mercurous tannate (0.1 gram thrice daily in pill form) or yellow mercurous iodide (0.01 to 0.02 gram thrice daily in pill form). Mergal in doses of three to six capsules daily is also valuable. Mergal is mercury cholate with tannin albuminate, and seems to cause no colic or renal irritation even when given for a prolonged period. Its action is mild and should be used

when vigorous treatment does not appear essential. In those malignant cases of syphilis which tend to cause rapidly destructive lesions and resist ordinary treatment, Wechselmann believes that calomel is the best remedy. It should be injected in doses of 0.1 gram, and the risk of poisoning must be assumed. In some isolated cases, the use of atoxyl has increased the patient's strength and weight so that he could tolerate a course of mercury treatment. Wechselmann says that a rapid method for securing the action of iodine is by the injection of iodopin. For several days 10 to 20 grams of warmed 25 per cent. iodopin solution is injected under the skin of the back or buttock.

The Use of Arsacetin in the Treatment of Syphilis.—NEISSER (*Deut. med. Woch.*, 1908, xxxv) believes that none of the newer remedies will replace mercury in the treatment of syphilis, but that they will often be of value to supplement the action of mercury or they may be used when mercury is not tolerated. He speaks of both the curative and prophylactic use of atoxyl, and then proceeds concerning the new arsenical preparation arsacetin introduced by Ehrlich. He believes that, of the two, arsacetin is much less apt to cause toxic symptoms and that the curative effects seem to be greater. The solution does not decompose, and can be sterilized. The initial dose should be 0.05 to 0.1 gram, which is gradually increased. Degenerative changes of the heart or kidney increase the liability to the production of toxic symptoms. In general arsacetin should not be given when there is myocarditis, and if the kidneys are insufficient, the urine must be frequently examined and the drug stopped if there are signs of kidney irritation. Some cases of kidney disease (possibly when due to the syphilitic virus) are benefited by its use. Neisser thinks that the treatment should be begun as soon as the diagnosis is made, either by finding the spirochetes or by the serum reaction. He believes that a combination of the arsacetin and the mercurial treatment will secure the quickest and best results. Arsacetin (0.6 to 0.75 gram) is injected on two succeeding days, and after an interval of four days this double injection is repeated. The entire treatment takes ten weeks, and 12 to 14 grams of arsacetin are used in all. Neisser used a 10 to 15 per cent. solution, which caused neither infiltration nor abscess. Mercury was given by inunction or by injection of gray oil. In some cases he also gave arsacetin 0.12 gram a day by mouth, but thinks that this method and dosage have not been sufficiently established.

Active Immunization by Subcutaneous Injections of Living Typhoid Bacilli in Typhoid Fever.—PESCAROLO and QUADRONE (*Zentralbl. f. inn. Med.*, 1908, xl, 989) present a preliminary report of their results with the use of subcutaneous injections of living typhoid bacilli. Their conclusions are as follows: (1) In typhoid fever, bacteriotherapy has a real value chiefly because of a stimulating action on the production of immunizing substances in the blood. (2) It is entirely harmless and causes only transitory symptoms of a local or general reaction. (3) It is of especial value in cases of typhoid septicemia, when the symptoms of a general infection are the chief characteristics. (4) The injections should be made early but not before the bacteriological diagnosis is made. The bacteriological diagnosis is made by the blood culture method.

PEDIATRICS.

UNDER THE CHARGE OF

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The Treatment of Otitis Media Complicating Scarlet Fever and Measles.—E. P. FOWLER (*Amer. Jour. Obst.*, 1908, lviii, 530) states that acute otitis media occurs probably in 20 per cent. of scarlet fever cases and in 5 per cent. of measles; the supervention of chronic suppurative otitis media he considers usually the result of neglect except in the tuberculous and other weak patients, in whom it may be chronic from the outset. Adenoids and tonsillar hypertrophy are a very important cause for the high percentage of cases of ear disease in measles and scarlatina; these conditions, therefore, should be corrected before children are taken with the exanthems. The preventive treatment is to care for the infections by keeping the room temperature at from 60° to 65°, the air moist so that the respiratory mucous membranes may not be irritated and the patient may not take cold in addition to the disease. Measles patients should be kept in bed for ten days, scarlet fever patients for twenty-eight days. To the neck should be applied a moist compress covered with rubber tissue. The mouth, nose, throat, and eyes should be kept clean, the nose being frequently cleaned of its secretions, one nostril at a time being compressed. The Politzer bag should be used gently, borovaselin for the coryza and sneezing, cocaine or weak adrenalin tampons for the catarrh. The throat should be swabbed regularly. These measures will prevent many ear and throat complications.

Should otitis develop, the bowels must at once be opened, diaphoresis produced, and the ear canal irrigated with either hot bichloride, boric acid or normal salt solutions, using two quarts of fluid every two hours; a suction ball instrument is the best instrument to use. As soon as pus is diagnosticated, an incision must be made through the tympanic membrane. Doubtful cases should always be incised after two to three days, also cases with slight bulging of the tympanic membrane with tenderness over the mastoid. In the presence of streptococci and mastoid tenderness, the mastoid should at once be opened to prevent meningeal involvement. Drainage must be resorted to and suction used at the dressings to bring all purulent matter to the surface.

Paralysis in Infancy due to Gastro-intestinal Intoxication.—H. LONGO (*Ann. de chir. et d'orth.*, 1908, xxi, 165) reports the case of a girl, aged four and one-half years, who had been twice paralyzed in the short course of her life. She was perfectly well and had developed normally until her ninth month, when an enterocolitis made her ill for ten days. This left her with constipation, for which enemas were employed. At thirteen months, while apparently in the best of health, she fell over and could not be made to stand again. Fever and drowsiness existed, but no vomiting or convulsions. A purge, milk diet,

and cold to the head caused a disappearance of the symptoms. A left-sided paralysis was noted, the mouth being drawn to the right. Absolutely all signs of this paralysis had disappeared by the eighteenth month. At three years the child again had an intestinal infection: constant slight fever, fœtid diarrhœa. This continued for a long time, the diarrhœa alternating with a persistent constipation; the tongue was constantly coated. Suddenly one day she again fell over and had a slight convulsive seizure, followed by somnolence, fever, and irritability, and a complete right-sided hemiplegia. Purgatives and enemas soon cleared up the patient's mental state, but the paralysis and aphasia persisted. After several weeks the paralysis disappeared and speech returned. The urine during the entire attack remained abundant, and contained no albumin. Four months after the onset not a sign of paralysis could be observed. In Longo's opinion both paralytic attacks were due to an intestinal intoxication. He excludes hysteria chiefly because of the age of the child; the functional nature of the condition is ably defended.

Hemorrhoids in Infants.—After reviewing the scanty literature on this subject, D. G. Zesas (*Arch. gén. de chir.*, 1908, ii, 355) reports such a case in a child aged three and one-half years. He finds that heredity is of importance in the development of hemorrhoids in infants. Any condition interfering with the circulation, just as in adults, may induce hemorrhoids; constipation plays a very important part. Other causes are tumors within the abdominal cavity, enlarged mesenteric glands, diseases of the liver, etc. Infections about the anus may cause a phlebitis, and a secondary hemorrhoidal state may result. Phimosis has been followed by hemorrhoids. Zesas' patient had been rachitic since birth; he had marked bleeding from the anus with each stool since his sixth month, and of late discharged blood even without stool. The cause of the condition has not been recognized. A Whitehead operation was performed and the patient recovered completely. The hemorrhoids may be in the form of an angioma or a simple varicosity. Zesas states that there should be no difficulty in differentiating it from rectal polyps, the only condition with which it might be confounded. The treatment should always be operative; recurrences have never been noted.

Artificial Infant Feeding.—Pisek (*Amer. Jour. Obst.* 1908, lviii, 694) summarizes this important subject for the general practitioner in the following sentences: All infants require a liberal supply of fats and carbohydrates to supply energy and heat, and a small supply of proteids and mineral matter to replace the daily loss. If these are supplied, the infant can get along for considerable periods of time without showing bad effects, but successful development and growth cannot take place. For proper growth, a liberal supply of proteids and mineral salts in addition to the quantity needed to replace waste is absolutely essential, for while gain in weight may result from the conversion of fats and carbohydrates of the food into body fat, growth or the formation of blood and tissue cannot occur unless there is more proteid and mineral matter in the food than is needed to replace loss. The development of the infant rests on the proteid supply. A portion of the proteids of

the food for healthy infants must be in the form of milk, as this is changed in the stomach, by the gastric secretions, into a semisolid food which is the forerunner of solid food. All infants conform to the general laws of nutrition, and no infant is a law unto itself except in nonessentials and in its preferences for different forms of food. Varying the form in which the food elements are presented has much to do with success in feeding, and feeding in difficult cases depends absolutely upon it. Infants differ to a marked degree in capacity for digesting and assimilating food. Some will be able to thrive and grow on a quantity of food on which other infants will not much more than hold their own. As each element of the food performs a special function in nutrition, it is important to know approximately the composition of all feeding mixtures employed, for excess or deficiency of one or all of the ingredients is attended with harmful results, if continued for any length of time. The raw materials for making up food for infants under all conditions consist almost exclusively of cow's milk, milk sugar, cane sugar, and the cereals. The successful infant feeder is the one who can combine these substances in such a manner as to meet the peculiarities of each particular infant.

Tabes and Juvenile General Paralysis through Acquired Syphilis.—APERT, LEVY-FRAENKEL, and MENARD (*Arch. de méd. des enfants*, 1908, xi, 477) report the case of a girl, aged fifteen years, whose father contracted syphilis when she was two years old; immediately after acquiring it he infected his wife. Both of them had initial lesions and a number of secondary manifestations, and treatment in the form of van Swieten's fluid was carried out but a short time. The father developed tabes six years later, and died of general paralysis twelve years after the onset of the syphilitic infection. The mother is living; she has tabes also, developing it about the same time as her husband. An older brother is living and well. The patient slept with her parents (between them) at the time they had their initial lesions. The child developed a purulent discharge, had ardor urinæ, and lost much flesh. She also had a cutaneous eruption some weeks later. At the age of fourteen there was headache, vomiting, fever, and coma, all of which disappeared after some days. Her present illness was ushered in by an epileptiform convulsion, during which she had conjugate deviation, nystagmus, vomiting, positive Babinski, and absence of knee-jerks. The latter persisted after the attack; there is also now incontinence of urine and attacks of vomiting. After recovering from the acute attack the child remained quiet, melancholy, and somewhat queer. The pupils became unequal, the Argyll-Robertson sign developed, and later also an iridochoroiditis. A trophic ulcer has made its appearance on the outer side of the foot. Romberg's sign is positive and the gait somewhat characteristic of tabes dorsalis. There is no disturbance of speech or writing. The mental traits of general paralysis have become more and more pronounced. There is, however, no tremor, speech- or writing-disturbance. Antisyphilitic treatment has been of no avail.

OBSTETRICS.

UNDER THE CHARGE OF

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Elongation of the Cervix Uteri Complicating Labor.—ROUVIER (*L'Obstétrique*, August, 1908) draws attention to this condition as a complication of labor. He has observed 20 cases, and finds the cause to be some obstruction in the circulation of the uterus, arising during labor. In primiparous patients this condition arises when the head is pressed deeply down into the pelvis and remains there some time with full dilatation. In extreme cases the enlargement resembles a polyp, and is usually upon one side of the cervix only. In multiparous patients one might expect this condition more frequently, because of the progressive increase in the size of the fœtus. Statistics, however, do not bear out this hypothesis; most of these cases occur when the fœtus is normal in weight. The condition has been observed in premature labor. In multiparæ the important factor in producing this complication is absence of flexion in the fœtal head, or the developing of some unusual presentation. In twin pregnancy, when dilatation proceeds very slowly, this condition develops, but never to an extreme degree. In some primiparæ it has been observed accompanied by pelvic deformity. In most cases the tumor resembles somewhat in shape the mouthpiece of a flute. When swelling is extreme the tumor may be present in the vagina. In diagnosis it is to be distinguished from uterine polyp attached to the body of the womb. The elongation resembling a polyp is never present excepting during labor, while a chronic condition of polypoid degeneration may be found indefinitely. This enlargement of the uterine neck usually disappears spontaneously after labor, while a uterine polyp would remain. The condition may also be confused with cystocele or rectocele, which is greatly exaggerated at the moment when the fœtus is expelled. It may also resemble a vascular polyp, for which the operation of extirpation may be undertaken. Fibroid tumor developing from the neck of the uterus should not be mistaken for this condition, if the pedicle of the tumor can be recognized. If the placenta should present in this position, it would become more or less separated during labor, resulting in hemorrhage, which should lead to diagnosis. The prognosis of unilabial œdematous enlargement of the cervix varies in accordance with a number of factors. When a diagnosis is made early and labor is conducted in accordance with the symptoms which develop, the prognosis is favorable. The spontaneous resolution of the tumor during labor is also advantageous. In rare cases the separation and expulsion of the tumor before the fœtus may be distinctly favorable. The formation of a thrombus or hematoma is an undesirable complication. This may rupture during labor or as late as the fourth or fifth day after. So far as the fœtus is concerned, it is not exposed to additional risks so long as the membranes are unruptured. After the amniotic liquid escapes the cervix may increase the pressure upon the fœtal head.

In dealing with these cases one must not expect the spontaneous disappearance of the tumor during labor. In premature labor with the membranes unruptured, interference should be avoided. The patient should be kept at rest and given sedatives. After the rupture of the membranes, especially at term, the case should be brought to an end. The occiput should be rotated anteriorly, if possible, and the swollen portion of the cervix pushed upward and released from pressure. It is occasionally necessary to make small multiple incisions into the cervix to release imprisoned blood and serum. When the pelvis is normal and labor delays, the child may be extracted with the forceps. If pelvic deformity of moderate degree is present, a better result is usually obtained by version. In the puerperal period this condition usually disappears from the ninth to the thirteenth day. Should the tumor remain it may be finally removed.

Perforative Appendicitis Complicating Pregnancy.—BABLER (*Jour. Amer. Med. Assoc.*, October 17, 1908) reports his collection of 103 cases of perforative appendicitis complicating pregnancy. The etiology of the disorder in pregnancy does not differ from that in non-pregnant patients. Three-fourths of the cases develop after the third month of pregnancy. Perforation occurred in 44.6 per cent. of cases. Portal infection rarely follows. The uterus may become infected through the peritoneum, through the lymph and bloodvessels, or through adhesions from the abscess wall to the pelvic organs. In many cases the pregnant uterus forms a part of the abscess wall. Rupture of the abscess follows contraction of the uterus and expulsion of the fœtus. In some cases abscess of the appendix may form an obstacle to delivery. Diagnosis is usually made without difficulty, although in some cases ruptured tubal gestation may be confused with appendicitis. When perforation occurs, the mother's mortality averages 48.5 per cent., the fœtal mortality 66 per cent. In the 103 perforative cases collected, operation was performed in 89, followed by abortion in 37; 36 mothers died. There were 14 perforative cases treated medically, all of which died; 10 of these aborted, and in 10 the child died in utero, making a maternal mortality of 100 per cent. and an infant mortality of 75 per cent. Of the 104 non-perforative cases, 50 were operated upon, 7 aborted, one mother died. Of the 54 not operated upon, 6 aborted, 4 mothers died. During the first ten days of the puerperal period 28 cases of appendicitis were collected, of which, 18 were perforative; 12 of these women were operated upon, with a mortality of 33.3 per cent.; 2 of the 6 not operated upon recovered by accident: the pus burrowed into the rectum. Nine cases were non-perforative, all of which recovered, whether treated by operation or not.

So far as treatment is concerned, these statistics strikingly emphasize the fact that operation offers the only hope of success. Operation must be done as early as possible. With perforation and localized abscess, incision and free drainage are indicated. If possible, the uterus should not be emptied before the operation, lest the diminution in its size rupture the wall of the abscess, causing pus to enter the general peritoneal cavity. If the patient has general peritonitis, incision and drainage, without disturbing the pregnancy, are indicated in early gestation. When the patient is near term the operator must decide between Cesarean section

or forcible extraction of the child followed by abdominal incision and drainage. The treatment of suppurative peritonitis is indicated in a general way.

Babler reports the case of a multipara six months advanced, who during pregancy had frequent pain in the right lower abdomen. This increased and fever developed with great tenderness over the appendix. The cervix was slightly dilated. The fœtal heart sounds were normal. The patient was brought some distance to a hospital, and upon making an incision over the area of dulness a quart of offensive pus was evacuated. The wall of the uterus formed a part of the abscess wall. The appendix could be felt near the ribs, curled on itself, perforated, containing a fecal concretion, which was so firmly adherent and so high in the abdomen that it was possible to remove only the distal, free portion. Rubber tubes were inserted for drainage, and gauze drains were placed between the abdominal wall and the uterus, so that the general peritoneal cavity might be protected if labor developed. Silkworm-gut sutures were placed in the upper and lower angles of the incision as a precaution. Two days after operation labor pains developed and a six months' fœtus was expelled. The patient made a gradual recovery, with a temperature reaching as high as 104° on several occasions. When the drains were removed the wound surface was found covered with a necrotic membrane. The general peritoneal cavity was walled off, and under irrigation of the wound twice daily the patient's temperature fell. She afterward developed phlebitis in the left thigh. Her final recovery was complete.

Perforation of the Uterus with Injury to the Intestine.—Ssadowski (*Zentralbl. f. Gynäk.*, No. 41, 1908) reports the following interesting case: The patient summoned a physician for profuse hemorrhage, occurring one month after an abortion. It was supposed that remnants of the ovum remained, and dilatation and curetting was performed. As nothing was removed by the curette, the placental forceps was inserted and brought away what was described as a mass of soft tissue. This was cut off and found to be the intestine. Consultation was immediately summoned and the patient transferred to hospital. The original curetting was performed in the patient's lodging, with the assistance of a midwife only, although the patient was placed across the bed and a speculum used. On admission to the hospital the patient had a normal pulse and temperature, although the pulse was weak. The abdomen was slightly distended and there was moderate hemorrhage from the vagina of dark blood. A piece of small intestine between 2 and 3 cm. long was hanging from the vagina. This could be traced to the uterus, which was found slightly enlarged and anteverted, with an elastic tumor on the left side. The abdomen was opened as soon as possible and the omentum found greatly injected. There was dark fluid blood in the abdominal cavity, mixed with masses of feces and having a strong fecal odor. The intestine was much distended. A rent in the uterus in its anterior wall showed where the instrument had passed through, and where a mass of intestine had been grasped and torn from its mesentery. There was an ovarian cyst as large as a fist on the left of the uterus. The mesentery was examined, and that which had been injured was removed. The ends of the intestine were freshened and brought together. A tear in the cecum

near the ileocecal valve was closed by suture. The uterus was amputated at the cerxix, the cervix cauterized, and the ovarian cyst removed. As the abdomen was considered infected from feces, the abdominal incision was left open. Gauze tampons were inserted wherever necessary to stop the oozing from the mesentery and provide drainage. For five days after the operation the patient had considerable fever and a pulse from 120 to 130. On the fourth day the bowels moved spontaneously and the patient had diarrhœa for a few days. On the forty-ninth day after operation the patient left the hospital with an abdominal fistula about one inch in depth. She returned afterward for outpatient treatment until she completely recovered.

GYNECOLOGY.

UNDER THE CHARGE OF

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Ultimate Results Obtained from Surgical, in Comparison with Medical, Treatment in Certain Ovarian Diseases and in all Ovarian Neuroses.—HODGES (*Virginia Med. Semi-Monthly*, 1908, xiii, 222) believes too little time and skill are usually extended by both physicians and surgeons in the proper diagnosis of cases of ovarian disease and ovarian neuroses. Critical consideration is required, not only of the special features of each case and the particular existing pathological condition, but also of the individual temperament in order to reach a correct diagnosis. The systemic condition of the patient should receive first consideration, inasmuch as the removal of a single pelvic abnormality does not often effect a cure. Meddlesome pelvic interference in the treatment of these cases is to be condemned. Certain ovarian diseases and most ovarian neuroses do not usually require operative measures. Frequently operations on this organ are too radical and are performed before medical measures have been given a full and fair trial. Oftentimes by such operations an anatomical but not a physical and psychical cure is produced. Hodges offers a plea for harmonious coöperation between the surgeon and the physician in the treatment of such patients.

Primary Carcinoma of the Fallopian Tube.—GEMMELL (*Jour. Obst., and Gyn. Brit. Empire*, 1908, xiv, 31), in reporting a case of primary cancer of the Fallopian tube, states that cancer of that structure, either primary or secondary, is of rare occurrence, the case of Orthmann, in 1888, being the first one reported of the primary variety. In 90 cases of hysterectomy for cancer of the uterus the appendages were found involved secondarily in three instances and in 12 cases of ovariotomy for cancer of the ovary no instance of involvement of the tube was noted. Gemmell believes that a predisposition to cancer of the tube lies in inflammatory changes and that a coincident papillomatous growth of

the tubal mucosa paves the way for the change to malignancy. The occurrence of the disease in the middle and external portions of the tube indicates that it is a sequel to inflammatory trouble. Macroscopically it is difficult to distinguish between exuberant inflammatory product and newgrowth, and the aid of the microscope is necessary in deciding this point. The disease is generally unilateral and commonly limited in area. The clinical picture is usually that of a woman, aged about forty-five years, who has suffered from long-standing pelvic inflammatory troubles, and with some recent pelvic pain, with vaginal discharges, often profuse, sanious or watery, and some abdominal swelling. The pelvic tumor present rarely reaches above the pubes, and ascites is not always present. Recurrence and metastases are said to be rapid and fatal, yet Boxall recorded a case of carcinoma of the tube, secondary to carcinoma in the ovary, in which there was no recurrence five years later.

Late Recurrence of Mixed Ovarian Cysts in Spite of Extirpation.—VINCENT (*Ann. de gyn. et d'obst.*, 1908, v, 478) reports three cases in which he had extirpated ovarian tumors of a mixed character, that is, solid and cystic, and had been surprised to note that the return of the growths, malignant in nature, was delayed from two to nine years. Vincent suggests that the exposure of the involved tissues to air has an effect nearly as salutary as to peritoneal tuberculosis.

Gynecological Massage in the Treatment of Plastic Infiltrations and of Pelvic Exudates.—BOURCART (*Ann. d. gyn. et d'obst.*, 1908, v, 449) has used massage twenty years in his gynecological work, and concludes that massage belongs to the medical treatment and will give excellent results, especially if used reasonably and prudently and with a good technique. It should not be employed exclusively. He warns against its use in the active stages of pelvic infections, and suggests that bimanual massage be not employed in inflammatory condition of the appendages.

Infection of Operative Wounds by Malignant Disease.—I. S. STONE (*Jour. Amer. Med. Assoc.*, 1908, li, 1748) reports a case in which carcinoma or sarcoma very rapidly manifested itself in the sides of the abdominal wound a few weeks after a section had been made for cancer or sarcoma of the pelvic structures, following vaginal hysterectomy by two years. He recommends careful protection of the operation field against malignant infection.

The Principles upon which the Success of Surgical Treatment of Retrodisplacements of the Uterus Depends.—R. C. COFFEY (*Surg., Gyn., and Obst.*, 1908, vii, 383) recommends an operation consisting of plications of the round ligaments and adjacent parts of the broad ligaments on to the anterior surface of the body of the uterus for the relief of retrodisplacements of that organ, and bases it upon the following principles: (1) The general scheme of nature is to hang all abdominal organs by peritoneal ligaments; (2) these ligaments are strong enough to hold several times the weight of the organs they are intended to support and notwithstanding the fact that the normal resiliency of the various organs probably aids materially the peritoneal ligaments to determine the position of the organs; (3) the peritoneum is loosely attached to

the abdominal walls, being firmly fixed only at the diaphragm, thus permitting displacements of viscera in degrees varying according to the distance from the diaphragm; (4) the peritoneum is ideally adapted for making new ligaments, inasmuch as when two peritoneal surfaces are brought together and held firmly in an aseptic state they adhere, blend, and obliterate, and the contiguous surfaces lose their endothelial covering and became continuous at the point of peritoneal union; (5) that muscle fibers are found only where intermittent motion is needed; (6) the uterus is partly abdominal and partly extraperitoneal, its abdominal ligaments (broad) being attached near its middle, thus giving it a pivotal hanging with the heavy end above the pivot; (7) muscular ligaments act independently of the peritoneum and cannot be made to adhere permanently to its connective-tissue side; (8) a large amount of connective tissue exists near the round ligament, making folding of this ligament with the broad ligament advisable to add strength; (9) ventrosuspension or ventrofixation of the uterus makes useless the round ligaments. Coffey insists that the ideal operation for retrodisplacements of the uterus consists in some form of operation which shortens the anterior fold of the broad ligament, at the same time temporarily utilizes the round ligament for its accompanying connective tissue, but which uses absorbable sutures and thus makes provision for complete recovery of the position and function of the round ligaments as soon as the sutures are absorbed.

Removal of an Ovarian Dermoid Cyst Complicating Pregnancy and without Disturbing the Pregnancy.—STROTHER (*Virginia Med. Semi-Monthly*, 1908, xiii, 259) relates a case of dermoid cyst of the ovary complicating pregnancy of three months. The tumor was removed by abdominal section and the pregnancy was not interrupted.

Early Sitting Up and Rising after Laparotomy.—An editorial (*Lancet*, 1908, i, 1225) comments on the contrast between the old plan of keeping laparotomy patients in bed four weeks after operation, and the new one of allowing such patients to sit up in one day. Ries, of Chicago, was the pioneer of the latter plea, he having employed it for six years, and Boldt for a shorter period of time. Boldt has brought the matter to the profession by his writings. In England, Chandler has employed it in 200 cases. He applies a single broad band of zinc oxide plaster, reaching from the umbilicus to the pubes, after closing the abdomen. After recovery from the effects of the anesthetic the patients were kept, by means of a bed-rest, in a sitting posture for twelve hours and then allowed to change to lying and sitting positions as they wish. On the third or fourth day they sit in a chair and all walk on the fifth day after operation, leaving the hospital well and strong from the tenth to the fourteenth day.

The Causes of Inversion of the Uterus.—ATTHILL (*Brit. Med. Jour.*, 1908, i, 1224) believes most cases of inversion of the uterus do not occur in the puerperium as generally believed. He reports cases due to the presence of sessile tumors attached to the fundus and efforts at expulsion from the uterus pulled down and inverted the fundus uteri. He also believes that in puerperal cases inversion is due to attachment of the placenta to the fundus uteri.

OTOLOGY.

UNDER THE CHARGE OF

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Local Anesthesia in Otology and Rhinology.—HAYMANN (*Zentralbl. f. Ohrenheilk.*, 1908, vi, 203), in the general consideration of the subject, defines the uses and limitations of local anesthesia, both as to the means employed and the method of application, in the former drawing comparisons between cocaine, which it is desirable to replace by a less dangerous drug possessing equal anesthetic power, and alypin with one-sixth the danger of cocaine and such other of the numerously prepared substitutes as novocaine, tropacocaine, and eucaïne; in the latter distinguishing between anesthesia by superficial absorption and by infiltration, with a decided preference for the latter, because of its larger applicability and greater certainty of effect. Of the later proposed substitutes for cocaine, Haymann gives the preference to alypin and novocaine; the former for superficial application, to mucous surfaces for instance, and the latter for infiltration, both preferably combined with adrenalin which decreases the irritative effect of alypin and increases and prolongs the otherwise comparatively transitory anesthetic effect of novocaine. Aside from slight invasions of the auricle for which an ether spray affords sufficient local anesthesia, extensive incisions and dissections may be painlessly made after injections at two points: one close to the superior attachment of the auricle, and one below the lobule in the furrow between the mastoid process and the articulation; from these two puncture points the anesthetic solution may be injected subcutaneously around the attachment of the auricle and also carried inward, from the lower puncture point, along the wall of the external auditory canal.

While the intact epidermal lining of the external canal and outer coat of the drumhead afford little entrance to anesthetics locally applied, these structures possess, in part, so nearly the same nerve supply that anesthesia of the canal implies a measure of anesthesia of the drumhead.

Paracentesis of the drumhead, although a slight operation, is one demanding care in the choice of the point of invasion and in the carrying out of any extended incision; aside from the question of pain, anesthesia, therefore, is desirable. Iwanow reports a sufficient anesthesia for paracentesis, from a pad of cotton soaked in a cocaine-adrenalin solution and left for ten minutes in contact with the drumhead; other writers have attained the same end, in the same manner, with solutions of alypin of from 5 to 10 per cent., the local anesthesia being observably more rapid and more complete in proportion to the loss of the epidermal layer, an observation in accord with the use of mild corrosive agents, carbolic acid, for example, in conjunction with local anesthetics superficially applied.

For the more complete anesthetizing of the drumhead, as well as for operations within the middle ear, such as divisions of adhesions, curetting, removal of the major ossicles, and of the outer epitympanic wall, the procedure of Neumann has been found satisfactorily to answer the desired purpose.

Through a split or fenestrated speculum, an ether spray is thrown upon the superior canal wall at the point chosen for injection of the anesthetic solution, by means of a Pravaz syringe with a curved needle, the point of puncture being the junction of the cartilaginous and osseous portions of the canal, and the needle being carried slowly inward as close to the bone as possible, the injection of the solution covering the whole course of the introduction of the needle.

For still more extensive operative interference on the mastoid portion or for tympanomastoid exenteration, a system of infiltration anesthesia, as carried out by Neumann, has been found to be so far effective as to make it possible to dispense with a general anesthetic. The injections in these cases, made about fifteen minutes before the time for operation, and repeated during the operation if necessary are, for the mastoid incision, three in number, controlling the superior, the inferior, and the middle portions of the mastoid surface. They are made periosteally and in the direction of the incision in the soft tissues. These injections are followed by two entering at the posterior conchal fold and running inward parallel to the posterior canal wall, there being also an injection similar to that made for the purpose of removal of the outer epitympanic wall and other operations in the epitympanum; this combination of the subperiosteal injection of the mastoid with the injection in the canal is sufficient, according to Neumann, to afford a local anesthesia sufficing for the performance of the radical operation and, in fact, in forty radical operations done in Politzer's clinic, in which this method was employed, the resultant anesthesia was effective.

Notwithstanding the favorable reports upon which the author bases his article, he is of the opinion that more intimate observation upon a larger amount of operative material will be necessary to determine decisively the best means by which local may be substituted for general anesthesia in aural operations.

Influence of Pregnancy on Acute and Chronic Suppuration of the Middle Ear.—Ferreri (*Archiv. Italio di Otol.*, 1908, xix, p. 293; *Zentralbl. f. Ohrenheilk.*, 1908, vi, p. 322), in addition to various references to the observations of other writers upon the injurious influence of pregnancy upon otosclerosis, contributes his own observations in this respect, and also in reference to cases in which suppuration is a symptom, as, for example, the common affliction of pregnant women with furunculosis of the external auditory canal, and the fact that pregnancy has an unfavorable effect in the apparent prolongation of suppuration from the middle ear. Under these circumstances it is Ferreri's opinion that, in the event of the occurrence of an acute mastoiditis, operation should be as early as possible, the existence of a nephritis of pregnancy being no counterindication. In one instance under his observation the effect upon a chronic discharge of the ear on a developing pregnancy was so clearly determinable that the patient was first made aware of the existing pregnancy by the increase in the aural manifestations. Cases in which the unfavorable changes in the ear are evidenced during pregnancy present a more favorable prognosis than those in which the same influence is the result of lactation.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Experimental Production of Gastric Ulcer in Guinea-pigs.—STERNBERG (*Zeit. f. Heilk.*, 1908, xxviii, 280) has been able to produce ulcerations of the gastric mucous membrane of guinea-pigs, by feeding through a stomach tube, small quantities of alcohol. The lesion starts first as an area of necrosis covered with a slough, developing later into a deep circumscribed ulcer which extends to the muscular coat and heals gradually. The possibility that an abrasion of the mucosa, produced by the passage of the stomach tube, might be the cause of the ulceration was excluded by control experiments. The ulcers were usually situated about the cardia and not, as is the case in man, near the pyloric end. For the production of an ulcer, it is neccessary that the alcohol should come in direct contact with the gastric mucosa. This could only be accomplished when the animal's stomach was empty; when the stomach contained food the mucosa was protected and ulcers did not develop.

The Hemolytic Action of Bile and its Inhibition by Blood Serum.—It is known that bile has a toxic action upon at least two tissues of the body. It is hemolytic and directly injurious to the cells of the pancreas. In investigating the protective powers of the organisms against bile, SELLARDS (*Johns Hopkins Hospital Bulletin*, 1908, xix, 268) finds that normal blood serum protects effectively against the hemolytic action of bile salts in dilutions as high as 1 to 3000. Precipitation of the proteid by heat does not affect this inhibitory action. The fact seems to have a distinct bearing upon the tissue destruction in acute hemorrhagic pancreatitis following the injection of bile into the pancreatic duct. The bile itself causes a necrosis of the pancreas, but the hemorrhage would presumably tend to neutralize the action of the bile and thus prevent further necrosis. In simple catarrhal jaundice, except for the protective action of the blood serum, serious consequences, such as the development of a severe anemia, might result.

The Pro-infective (Aggressive) Action of Normal Blood Serum.—COLE (*Johns Hopkins Hosp. Bull.*, 1908, xix, 249), while studying the natural immunity of pigeons to Bacillus pneumoniæ, made some experiments to determine whether or not this natural immunity was dependent upon substances contained in the blood serum of the pigeon. By means of the technique employed in demonstrating the so-called Pfeiffer phenomenon, an effort was made to study the effect of combined inoculation

of pigeon's serum and emulsions of Bacillus pneumoniæ into the peritoneal cavity of mice. Into one series of mice 1 c.c. of normal pigeon's serum + 1 c.c. of emulsion of Bacillus pneumoniæ was injected intraperitoneally; into another control series of mice 1 c.c. of an emulsion of Bacillus pneumoniæ alone was injected. Fluid removed from the peritoneal cavity in the two series of mice after one hour showed no differences either as regards the type of exudate or the amount of phagocytosis. Attempts were then made to see if blood serum of pigeons and rabbits, when injected simultaneously with emulsions of Bacillus pneumoniæ, would protect mice against small doses of the organism, doses which under ordinary circumstances were just sufficient to kill. It was a great surprise to find that under these conditions the injection of pigeon's serum or rabbit's serum hastened rather than retarded the death of the animal. The result is quite contrary to what one would expect. The explanation for this phenomenon does not at first seem clear. It is possible that the foreign serum may form precipitins with the serum of the injected mice; that it may bind the complements of the injected animal, thus reducing the bactericidal property of the serum; or that the foreign serum may render the mice hypersensitive, thus setting up a state of conditions analogous to anaphylaxis; it is also possible that the "natural aggressins" described by Bail and others may be due in part to the injection of serum together with bacterial cultures.

The Effect of X-rays upon the Formation of Specific Anti-bodies.—Läwen (*Mitth. aus d. Grenzgeb. d. Med. u. Chir.*, 1908, xix, 141) has undertaken an extensive study of the effect of Röntgen irradiation in experimental infections, directing his attention particularly to its effect on the production of specific antibodies. Rats, mice, and rabbits were used in his experiments. Läwen details his experiments at considerable length and arrives at the following results: Long-continued exposure of animals to the *x*-rays with resultant destruction of the greater part of their leukocytes changes in no way the bactericidal power of the serum. The dissolution of the leukocytes adds no free bactericidal "endo-enzymes" which are capable of increasing the action of the serum alexines. The resistance of the animals exposed to the *x*-rays, on the contrary, was constantly found diminished for various bacterial inoculations. If the exposures had not been too prolonged, a leukocytosis followed the experimental injections just as in normal animals, the polymorphonuclears being particularly increased. After very long exposure to the *x*-rays the leukocytes gradually decreased in number, and finally disappeared completely from the peripheral circulation. While the Röntgen rays showed no action on the normal agglutinins, the formation of the specific agglutinins and to a much less degree of the specific bacteriolysins was greatly inhibited. In some animals the agglutinins were not formed at all. From this fact Läwen concludes that the hematopoietic organs are to be looked upon as the place of origin of the specific agglutinins.

Studies on the Relationship between Respiration and Blood Pressure.—Thomas Lewis (*Jour. of Phys.*, 1908, xxxvii, 213) finds that the heart is peculiarly susceptible to changes in pressure external to it. In cats a rise of intraperitoneal pressure of 1 mm. Hg. lowers systemic pressure

on an average of 8 to 9 mm. Hg. Conversely, a fall of pericardial pressure of 1 mm. Hg. raises systemic blood pressure by 8 to 9 mm. Hg. In small cats there is a rise of blood pressure accompanying suspended or prolonged inspiration, which is entirely due to the change of intrapericardial pressure. It is in no measure the result of decreased resistance to the flow through the lung vessels, and, indeed, it has never been conclusively demonstrated that changes in the diameter of the lung vessels resulting from alteration of intrapleural pressure, in natural or vagal breathing, has any appreciable influence as a factor in the production of the inspiratory rise of blood pressure. In large cats the same results could not be invariably demonstrated; nevertheless it seems certain that increased diastolic filling of the heart plays a large part in causing the inspiratory rise of blood pressure in these animals. Further experiments along this same line of investigation showed that in man a deep intercostal inspiration, which is not prolonged, yields a pure fall of blood pressure. A deep, diaphragmatic inspiration, which is not prolonged, gives a pure rise of blood pressure. The rise in blood pressure in abdominal breathing is due to a raised intra-abdominal pressure. In expiration the reverse is true. It is, therefore, not possible, either in man or animals, to state what the blood pressure response to a particular respiratory act will be, unless the conditions and nature of the act are known. Artificial respiration, as ordinarily employed, gives a rise of blood pressure during inspiration and a fall during expiration in all animals. Lewis believes that the pulsus paradoxus is a misnomer. Kussmaul's pulse is a normal event, for the blood pressure always falls when the patient, instructed to do so, takes a deep breath.

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